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THE PARATHYROID GLANDS IN HEALTH AND DISEASE.*†

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INTRODUCTION

Prior to the isolation of the active principle of the parathyroid glands,[1, 2] our knowledge concerning their physiological function was meagre. The many studies‡ since the advent of physiologically active extracts have not only increased our knowledge of parathyroid gland physiology but also our understanding of calcium and phosphorus metabolism in health and disease. In consequence, one can no longer consider the bony skeleton as representing a stable structure from the point of view of metabolism. The bony skeleton represents an active metabolic structure, influenced by many factors, the effects of which are readily demonstrated by histologic or metabolic changes.

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‡The author has not attempted to compile an inclusive bibliography of the vast literature pertaining to either calcium and phosphorus metabolism or the parathyroid glands in health and disease. In consequence, many important references are not included.

NORMAL CALCIUM AND PHOSPHORUS METABOLISM

The chief function of the parathyroid glands is the regulation of calcium and phosphorus metabolism. Therefore, an understanding of the basic concepts of normal calcium and phosphorus metabolism is a prerequisite to any discussion of the parathyroid glands.

The bones, composed chiefly of a complex calcium salt, containing calcium phosphate and carbonate ions, are the only storehouse of calcium and phosphorus in the body. As a system, they are perhaps best represented by the simple schematic drawing shown in Figure I. Here the bones are pre-

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sented by a bucket. The walls of the bucket represent the bone cortex, the sediment at the bottom of the bucket, the bone trabeculae. The bucket is filled with blood containing many other substances than calcium and inorganic phosphorus. However, the calcium and phosphorus are all that need concern us in this discussion. In normal individuals the concentration of these substances is extraordinarily constant. The serum calcium varies from 9.5 to

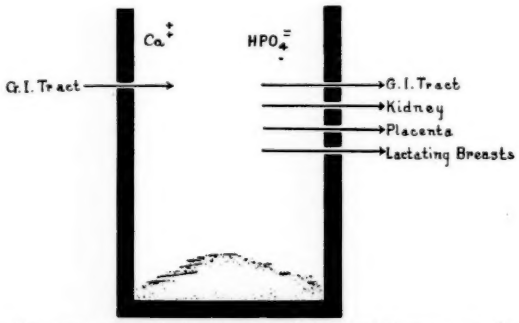

Fig. I.—A schematic drawing in which a bucket represents the bones as a system. The bucket is filled with blood and normally contains 9.5 to 11 mgm. of calcium and 3.5 to 4.5 mgm. of phosphorus per 100 c.c. The walls of the bucket represent the bone cortex, the sediment at the bottom, the bony trabeculae. The state of equilibrium existing in this system in respect to calcium and phosphorus is dependent upon the amount entering it via the gastro-intestinal tract and the amount excreted via the gastro-intestinal tract and kidneys. If entrance into the system exceeds the loss, a positive calcium and phosphorus balance exists, whereas, if the reverse is true, a negative calcium and phosphorus balance ensues. The loss from the system is further accentuated during the state of pregnancy or lactation. In such instances the placenta and lactating breasts serve as additional avenues of exit.

11 mgm. per 100 c.c. and the serum inorganic phosphorus from 3.5 to 4.5 mgm. per 100 c.c. The maintenance of this relative state of constancy of serum calcium and inorganic phosphorus is ample evidence that the bones are labile structures. Thus, in times of need, calcium and phosphorus are absorbed from the bones, and in times of excess deposited in bones. Certain experiments[3, 4] suggest that the bone trabeculae (sediment at the bottom of the bucket) serve as the most readily available storehouse of calcium and phosphorus and that the cortical bone (walls of the bucket) is at first spared in the process of mobilization. Thus, it would appear that in the adult, in times of need, calcium and phosphorus are first absorbed from the trabeculae and in times of excess are again deposited in the trabeculae. Such metabolic activity is readily demonstrated on histological examination of bones. Here, these two processes, bone absorption and bone deposition, are evidenced by increased cellular activity. Increased bone absorption is demonstrable by an increased number of osteoclasts and narrowing and reduction in number of trabeculae, whereas, with increased bone deposition, one observes an increase in the number and size of osteoblastic cells and an increased amount of osteoid tissue. Thus, from the degree and type of cellular activity observed in the skeletal system, one can estimate what metabolic changes are taking place. In normal bone, the absorption and the deposition of calcium and phosphorus go on continuously; consequently, there is always evidence of minimal osteoclastic and osteoblastic activity. Naturally, such cellular activity is more marked in the growing child. Whether the resorption and deposition of calcium and phosphorus are dependent solely upon the concentration of calcium and phosphorus in the blood cannot be answered with absolute certainty, although considerable evidence has been brought forth in favor of such a theory.[5] Adherents of this theory reason that under-saturation of calcium and phosphorus in the blood, as occurs in calcium and phosphorus deficiency diseases, calls for a liberation of calcium and phosphorus from the bones in an attempt to maintain a state of constancy in the blood.

On the other hand, in times of excess, the blood serum is to be regarded as a super-saturated solution of calcium and phosphorus. Under such conditions, the blood readily gives up its surplus and thereby enables deposition to take place. This state of super-saturation and consequent deposition of calcium and phosphorus is brought about in any portion of ossifying bone as a result of an enzyme called phosphatase.[6] This enzyme is secreted by osteoblasts and hypertrophic cartilage cells as well as by body tissues. This enzyme, phosphatase, is capable of hydrolyzing the phosphoric esters of the blood, such as glycerophosphate and hexosephosphate. In any ossifying area, the increased secretion of phosphatase allows for an increased hydrolysis of the complex phosphate compounds and in consequence a local increase in the concentration of the phosphate ions. This local increase results in a super-saturation of the blood with calcium and phosphate ions, the solubility product of tertiary calcium phosphate is exceeded, and, in consequence, deposition of this salt in the area of ossification takes place. The normal value for serum phosphatase is 2 to 4 units per 100 c.c. As one might expect, this value is higher in growing children or

any skeletal disease in which increased osteoblastic activity occurs.

One must next consider the many factors which influence the entrance and exit of calcium and phosphorus from this system because its state of equilibrium is dependent upon the balance between the intake and output of these two ions. If the intake exceeds the output, then calcium and phosphorus are retained and a positive balance ensues, whereas, if the reverse is true, a negative balance results.

Entrance into the system is dependent upon absorption of these elements from the gastro-intestinal tract and the amounts absorbed are in turn dependent upon:

1. The composition of the diet.[7]
2. The acidity of the gastro-intestinal tract.[8, 9]
3. The intestinal rate.[10]
4. Fat digestion and absorption.[10, 11]
5. Vitamin D.[12, 13]

Of these various factors, the diet and vitamin D are probably the most important. The daily calcium requirement for an adult is between 0.4 and 1.0 gm. Probably 0.5 gm. is adequate for the average individual. Equally as important as the calcium and phosphorus content of the diet is the proportion of the one to the other, the normal ratio of calcium to phosphorus being 2 to 1. For instance, if phosphorus is greatly in excess of calcium, the calcium may be precipitated in the bowel in the form of the relatively insoluble salt, tertiary calcium phosphate, and, in consequence, not be absorbed. Absorption of the remaining phosphorus is of no avail to the system because there is no corresponding amount of calcium available for deposition. In consequence, the phosphorus is excreted. Under such conditions, the result would be the same as if the diet had been inadequate in respect to both calcium and phosphorus. It is conceivable that the same state of affaris might exist if the calcium was greatly in excess of the phosphorus. Excess of certain mineral constituents may likewise be detrimental. An acid diet may favor calcium and phosphorus remaining in solution and thereby facilitate absorption. However, of all these factors, the calcium and phosphorus content and the ratio of calcium to phosphorus contained in the diet are the most important.

The acidity of the gastro-intestinal tract must influence the ionization of the calcium compounds present in the food and thereby increase their absorption. Unfortunately, there exists relatively little data enabling one to make a comparison of calcium and phosphorus absorption in individuals with and without achlorhydria.[9]

Any condition resulting in an increased intestinal rate would not allow adequate time for complete absorption of the ingested calcium and phosphorus. Therefore, we find evidence from time to time of calcium and phosphorus deficiency in diseases with associated chronic diarrhea, such as sprue, ulcerative colitis, etc. Bowel wall changes in such diseases may also be a factor preventing adequate absorption of these elements.

Failure to absorb fat, whether due to incomplete digestion or simply interference with absorption, results in deficient calcium absorption. Deficient absorption in such instances is due in part to the formation of relatively insoluble, non-absorbable calcium soaps. However, the chief factor is the failure to absorb the fat-soluble vitamin D, which is probably excreted with the excess fat. In such instances, there is an adequate absorption of phosphorus, but as this is not accompanied by calcium, it cannot be used for bone deposition and therefore is excreted in the urine. Such alterations in calcium and phosphorus metabolism eventually result in calcium and phosphorus deficiency disease. In the clinic, this type of disorder is seen in patients with chronic idiopathic steatorrhea or Gee's disease. It is probably due to a decreased or altered external pancreatic secretion as evidence by either low or absent lipolytic activity in the duodenal juice.[9]

Recent work with vitamin D[9, 12, 13] has shown that this substance influences calcium and phosphorus metabolism by increasing the absorption of calcium and phosphorus from the intestinal tract. Its chief role is that of regulating the normal functioning of the mucous membrane of the small intestine.

If one of the above-mentioned factors is at fault for an indefinite period of time, then absorption of calcium and phosphorus from the gastro-intestinal tract is interfered with, and, in consequence, entrance of these elements into the system does not take place. Thus, the equilibrium of the system is upset and there results a negative calcium and phosphorus balance. Long duration of this type of calcium and phosphorus metabolism disturbance is responsible for the calcium and phosphorus

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deficiency diseases, rickets and osteomalacia. In either condition, the infantile type of tetany may develop. The blood chemistry reveals a reduction of either the serum calcium or the inorganic phosphorus. In the severe types, both are reduced. The serum phosphatase is increased, indicating increased osteoblastic activity in this type of calcium and phosphorus disorder.

As can be seen from the diagram, there are four possible avenues of exit from this system. Under normal conditions, we need only to consider the loss which occurs via the gastro-intestinal tract or the kidneys. On a normal diet, the fecal calcium consists of both the unabsorbed dietary surplus and that which has been re-excreted into the bowel.[14] The urinary excretion of calcium varies from 0.1 gm. per day on a low calcium diet to 0.5 gm. on an adequate diet. Increased excretions by these avenues of exit occur in atrophy of disuse and in some cases of osteitis deformans. Such increases are, however, slight as compared to the increases observed in acidosis, hyperthyroidism and hyperparathyroidism. If the increased excretions exceed the intake, we have a resulting negative calcium and phosphorus balance. Such a state of affairs lasting for a long period of time will result in marked generalized decalcification.

Acidosis,[15, 16] whether due to disease or induced by the administration of large amounts of acids or acid salts, results in the excretion of large amounts of calcium and phosphorus. This increased excretion is solely via the urine and occurs whether the diet is adequate or inadequate in respect to calcium. Such increased excretions are not associated with increases in the serum calcium or phosphorus; in fact, if the subject has been on a low calcium diet for a long time, the administration of large amounts of ammonium chloride may cause a fall in both the serum calcium and inorganic phosphorus.[15]

Increased excretion of calcium and phosphorus far in excess of that resulting from acidosis or excess parathyroid extract has been observed in severe hyperthyroidism.[17] In such instances, the increased excretion is via the gastro-intestinal tract as well as the kidneys. Such increased excretions are observed in all types of hyperthyroidism as well as normal individuals receiving thyroid extract. With the institution of appropriate treatment for the hyperthyroidism, the excretions return toward normal as the patient improves. With controlled roentgenograms, one can demonstrate marked generalized decalcification in many patients with exophthalmic goitre.[17] This generalized decalcification is evidently due to osteoclastic lacunar absorption. Therefore, attention should be paid to the diet in this disease. The reverse situation is demonstrable in myxedema,[17] the average calcium and phosphorus excretion being 40 per cent below normal. Administration of thyroid to such patients results in an increase of both the metabolism and the excretion of calcium and phosphorus. Such studies would seem to indicate that thyroxin has a direct effect on bone katabolism. In neither hyperthyroidism nor myxedema is the serum calcium or serum phosphorus altered. Such findings demonstrate quite clearly that the height of the stream is no indication of its direction of flow into or out of the bones.

That the parathyroid hormone causes an increased excretion of calcium and phosphorus and, in consequence, generalized decalcification has been demonstrated by many observers.[18, 19] However, the exact mode of action is still the subject of some dispute. The dispute concerns whether the increased cellular activity of bone is a primary or a secondary effect. Collip and co-workers,[20, 21, 22] working with rats, consider the appearance of large numbers of osteoclasts as the first reaction, and therefore suggest that the hormone acts directly on the bone cells, stimulating them to release calcium and phosphorus. Albright *et al*,[18, 23] from observations made on humans, interpret the bone changes and resulting increased calcium and phosphorus excretion as secondary to the primary effect on phosphorus excretion. Thus, when parathormone is administered, the first metabolic changes are a rise in the urinary phosphorus excretion and a fall in the serum inorganic phosphorus. When parathormone is discontinued, the converse is true. This implies that the extra phosphorus is drawn from the body fluids. In such instances the changes in the rate of calcium excretion and in the height of the serum calcium are more sluggish and lag behind the phosphorus changes.

Because of this drop in the serum inorganic phosphorus, the equilibrium in our system is disturbed. In an attempt to maintain its normal state of constancy, the bones try to release phosphorus, but in doing so must also release the calcium deposited with it. In consequence, the serum calcium

rises, the urinary calcium excretion is increased, and the low phosphorus and increased phosphorus excretion continue. At this point, one observes evidence of increased cellular activity in the bone.

With large doses of parathormone, Collip[2, 24] produced a state of hypercalcemia in dogs (20+ mgm. per 100 c.c.). Once the serum calcium rises above 15 mgm. per cent, the inorganic phosphorus of the serum rises abruptly and continuously until death ensues. When this stage is reached, there is evidence of failing kidney function as shown by the rising non-protein nitrogen and urea as well as suppression of urinary secretion. With this grade of hypercalcemia, associated metastatic calcification of the kidneys, heart, lungs and stomach readily occurs. Such complications may occasionally occur in the clinic in cases of long-standing hyperparathyroidism.

The discovery by Parhon and Urechie,[25] MacCallum and Voegtlin,[26] that tetanic seizures and a drop in serum calcium followed the ablation of the parathyroid glands first served to link these glands with calcium metabolism. However, this knowledge concerning parathyroid deficiency and its association with calcium metabolism in no way enlightened us concerning the possible existence of the clinical state, hyperparathyroidism. Not until the isolation of the active principle of the parathyroid glands by Hanson[1] and Collip[2] was the state of hyperfunction of the parathyroid glands looked for in the clinic. Since then it has been established that this disease syndrome is not a rarity.

Today, clinical cases typifying both hypoparathyroidism and hyperparathyroidism are not infrequently encountered. These two states of altered parathyroid function are perhaps best grouped as follows:

1. Hypoparthyroidism:
   a. Due to operative removal.
   b. Cause unknown, the idiopathic type.
2. Hyperparathyroidism:
   a. Due to focal hyperplasia (ademona or neoplasia) of one or more parathyroid glands.
   b. Due to generalized enlargement and hyperplasia of all parathyroid glands.

HYPOPARATHYROIDISM

Etilogy.—Hypoparathyroidism following operations on the thyroid was recognized as early as 1881 by Weiss.[27] Thus, the post-operative type of hypoparathyroidism, though rare today because of the improved surgical technique employed in thyroid surgery, is still encountered. It is due either to injury or resection of the parathyroid glands. The fact that it is often a mild disease of short duration allows one to conclude that in many instances a marked suppression of function must result from minor trauma or interference with the blood supply. Parathyroid deficiency due to diseases such as deep inflammation of the neck tissues, hemorrhages, tuberculosis, etc., is evidently rare. Spontaneous or idiopathic hypoparathyroidism, a condition analogous to myxedema,[23, 28] is rare, yet in the past ten years, we have seen five such cases in this clinic.

Symptomatology.—Irrespective of the cause of the state of hypoparathyroidism, the symptoms, signs and laboratory findings which characterize it as a disease entity are the same. The symptoms include various types of paraesthesiae, muscular pains and cramps, carpopedal spasm, laryngysmus (which may be fatal), occasionally convulsions (often diagnosed epilepsy) and loss of consciousness. Any of these symptoms may be aggravated by exercise. In acute tetany such as occurs following a subtotal thyroidectomy, mental symptoms may appear early and are most disturbing to the patient. In such instances, the patient is unreasonable, extremely irritable, apprehensive of impending disaster and occasionally disoriented. The sudden fall in calcium to a low level may also cause visual symptoms. In chronic tetany one may note behavior changes, mental dullness and failure of memory for recent events. In chronic tetany, one may observe certain cutaneous changes, such as falling out of the hair, increased thickness and scaliness of the skin and marked changes in the nails with associated brittleness. Cataracts are frequent. A slit-lamp examination may be necessary to demonstrate the beginning lens opacities. One can also elicit a positive Chvostek's and Trousseau's sign and increased excitability of the nerves (Erb's sign). The laboratory tests reveal a low serum calcium and a high serum phosphorus. Roentgenograms reveal bones of normal density.

Diagnosis.—The other types of tetany must not be confused with parathyroid tetany. For instance, in patients with tetany associated with hyperventilation, excess of alkali, or loss of hydrochloric acid through vomiting, there is no change in the serum

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calcium, but a decrease in the degree of ionization due to the accompanying alkalosis and lowered pH of the blood. The infantile type of tetany is more apt to be confused with hypoparathyroidism, particularly the idiopathic type. The signs and symptoms are the same, but this type of tetany is always associated with a calcium and phosphorus deficiency disease such as rickets or osteomalacia. Here, the serum calcium may be lowered but the serum phosphorus is either normal or reduced and the X-rays show decreased bone density.

Treatment.—In the treatment of parathyroid tetany, one attempts to maintain the serum calcium near its normal level. If this is accomplished, the phosphorus falls and all signs and symptoms of tetany disappear. This can usually be accomplished by the administration of adequate amounts of the calcium salts and cod liver oil or one of the potent vitamin D concentrates. Restriction of the dietary phosphorus intake to about 1.0 gm. per day enables one to maintain a high calcium, low phosphorus intake and thereby insure the absorption of larger amounts of calcium unaccompanied by phosphorus. This allows for elevation of the serum calcium to take place without any appreciable rise in the serum phosphorus.

The amount of calcium salt administered and the number of times per day it is given are far more important than the particular salt employed. From the above, it is apparent that calcium phosphate should be avoided. One can employ calcium lactate in doses of 5 to 10 gms., dissolved in water, four to six times a day, depending upon the severity of the tetany. We prefer calcium chloride because it contains more calcium per gram of salt and because it tends to produce a slight acidosis which in itself is beneficial. We prescribe one-half to one dram of a 45 per cent solution of calcium chloride in a glass of water, four to eight times a day, depending on the severity of the tetany. This should be taken through a glass drinking tube. Cod liver oil (or its equivalent in the form of one of the concentrated preparations) in doses of 8 to 15 c.c. three times a day is sufficient to facilitate maximal absorption of the administered calcium salt. As the tetany improves, the amount of calcium salt and cod liver oil administered can be reduced, similarly the number of doses per day.

Thyroid[29] administered to patients with parathyroid tetany results in an increase in the serum calcium. Unfortunately, it cannot be used alone because the amount necessary to raise the serum calcium to a tetany-free level produces mild symptoms of hyperthyroidism. However, smaller doses in conjunction with the previously mentioned therapy may be employed, particularly in the rare post-operative cases having a co-existing thyroid deficiency. In uncomplicated parathyroid tetany, it is rarely necessary if ample amounts of calcium and cod liver oil are given.

Parathyroid extract (Hanson or Collip) is as a rule used only as an emergency measure because of the dangers of over-dosage, and the possibility of loss of effectiveness when used over a long period of time. Furthermore, parathormone injections are associated with considerable discomfort. Parathormone is also fairly expensive. In the rare case where it is a necessary aid, it should be given in doses of 5 to 10 units, once or twice a day. It should be discontinued as soon as possible.

During the acute distressing period at the onset of severe parathyroid tetany, more heroic measures may be necessary. If so, one can employ either parathormone intramuscularly or calcium chloride intravenously. If parathormone is used, it should be given in doses of 25 to 50 units every twelve hours. Prompt relief of laryngeal spasm, bronchial spasm or convulsions is obtained following the administration of 20 c.c. of a 5 per cent solution of calcium chloride. If given slowly, there are no untoward symptoms. It can be repeated three or four times in any twenty-four-hour period. In such cases, energetic administration of a calcium salt by mouth should be started as soon as the patient's condition has improved.

HYPERPARATHYROIDISM

Historical.—An adequate description of a previously unrecognized disease is often necessary to establish the fact that it is not a rarity. Such has been our experience with hyperparathyroidism, the first clinical diagnosis of hyperparathyroidism having been made independently by Mandl[30] in 1925 and by DuBois[31] in 1926. Mandl, acting on the suggestion of previous workers that generalized osteitis fibrosa cystica might be due to a parathyroid tumor, explored the parathyroid glands of such a patient, found a parathyroid tumor, removed it, and noted improvement. DuBois, studying a similar individual, noted the same alterations in the calcium and

phosphorus metabolism that had been observed in normal individuals receiving 100 units of a potent parathyroid extract and therefore concluded that generalized osteitis fibrosa cystica was due to a state of hyperparathyroidism. The number of cases reported since these epoch-making discoveries is sufficient proof that hyperparathyroidism is not a rare disease.

The two cases reported, one by Mandl, the other by DuBois, represent the classical form of hyperparathyroidism originally described by Von Recklinghausen.[32] This form of hyperparathyroidism should offer no diagnostic difficulties. It represents the end stages of hyperparathyroidism of long duration. Untreated, it is a progressive disease, which is usually fatal. Such cases are characterized by skeletal, renal, gastro-intestinal and constitutional symptoms. The metabolic alterations are hypercalcemia, hypophosphatemia, elevated serum phosphatase, hypercalcinuria, and hyperphosphaturia. The skeletal symptoms and changes are obvious, being manifested by pain, bone tumors, bone cysts, frequent fractures, disabling deformities, loss of height, etc. These are the tell-tale marks of the classical form of hyperparathyroidism or Von Recklinghausen's disease of the bone. Appropriate treatment at this stage of the disease will not permit complete restitution of the patient to a normal status because the skeletal changes are too marked and renal impairment too often present. Therefore, it should be our aim to make the diagnosis of hyperparathyroidism before such complications have occurred. In order to do this, one must acquaint himself with the varied nature of the presenting symptoms encountered in the other forms of hyperparathyroidism.[33] Then and only then will one be in a position to make the correct diagnosis as well as appreciate the fact that the most characteristic symptom may be closely simulated by other skeletal diseases.

Etiology.—The clinical entity, hyperparathyroidism, may be due to a hyperfunctioning adenoma of one parathyroid (neoplasia) or generalized hyperplasia of all the parathyroid glands.[33, 34] The localized tumor (adenoma or neoplasia) may involve a part of a gland, a single gland, or rarely parts of two glands. Hyperplasia is characterized by diffuse uniform involvement of all the glandular tissue. Such hyperplasia may represent the *wasser helle* or water-clear cell type and, occasionally, the chief cell type.

In speaking of hyperplasia of the parathyroid glands, one must appreciate that such can occur without any evidence of hyperparathyroidism. This type is known as functional or compensatory hyperplasia and may occur in long-standing calcium privation, rickets, osteomalacia, pregnancy and nephritis. In such instances, hyperplasia occurs because of the need for the hormone to maintain a normal serum calcium level, whereas, in hyperparathyroidism due to generalized hyperplasia of all parathyroid glands, there seems to exist some factor which drives all parathyroid tissue to produce more hormone than is needed, and, in consequence, osteitis fibrosa results. This type of hyperparathyroidism is analogous to the hyperthyroidism of exophthalmic goitre. Albright[35] suggests that this type of hyperparathyroidism may result from an increased secretion of the parathyreotropic hormone of the pituitary. The experimental work of Hertz and Kranes[36] and a review of the pathological findings in certain reported cases lends support to such a theory. The three cases of hyperparathyroidism due to hyperplasia of the parathyroid glands observed in this clinic were all marked cases exhibiting a hypercalcemia and hypophosphatemia responding to operative treatment.[35] They all had renal calculi (hyperparathyroidism with nephrolithiasis). The parathyroid hyperplasia in these three cases was of the water-clear cell type.

Calcium and Phosphorus Metabolism Changes.—The resulting increased production of the parathyroid hormone in either instance produces the same characteristic changes in the calcium and phosphorus metabolism observed in a normal individual receiving an active parathyroid extract.[21] Such alterations consist of:

1. An elevated serum calcium.* Values as high as 15 mgm. per 100 c.c. are not uncommon; values as high as 23.6 mgm.[37] per 100 c.c. have been reported. Any serum calcium value of 11 mgm. per 100 c.c. or over should be regarded as suspicious and therefore repeated.

2. A low serum phosphorus.* Values as low as 1.5 mgm.[33] per 100 c.c. have been observed. A

*All blood for serum analyses should be obtained fasting. Chemically clean syringes and centrifuge tubes must be employed. The serum calcium is done according to the Fiske and Logan[38] method, the serum phosphorus as described by Fiske and Subba Row,[39] the serum phosphatase after the method of Bodansky.[40]

serum phosphorus of 3.0 mgm. per 100 c.c. or lower, accompanied by an increased serum calcium, is almost always indicative of hyperparathyroidism because of the rarity of this combination in other diseases. The high serum calcium of multiple myeloma or metastatic malignancy is usually accompanied by a high serum phosphorus. The one exception to a high serum calcium, low serum phosphorus in hyperparathyroidism is observed in cases where, because of renal impairment, the serum phosphorus is elevated.

3. An elevated serum phosphatase.* This is demonstrable in all patients with hyperparathyroidism with detectable bone disease. As stated before, its serum level is directly related to the extent of the bone disease, particularly the degree of osteoblastic activity and independent of the degree of hyperparathyroidism. Therefore, in individuals with hyperparathyroidism without detectable bone disease, it is normal.

4. An increased calcium excretion. In an uncomplicated case, the increased calcium excretion is entirely urinary, the fecal excretion being unaffected. The calcium excretion may be increased three or four fold. In individuals with renal impairment, part of the increased excretion is fecal.

5. An increased phosphorus excretion. This is also entirely urinary. In the complicated cases, the urinary phosphorus excretion may not be affected.

Pathology.—The bones, being the only store-house of calcium and phosphorus in the body, are, in consequence, the source of the increased excretion of these elements. Unless the intake is equal to or in excess of the excretions, generalized decalcification will result. As Jaffee[41] states, an increase in the osteoclasts is the first histological evidence of the increased calcium and phosphorus excretion, the other bone changes occurring later. Being a disease of endocrine origin, hyperparathyroidism must exert its fundamental action, demineralization, on the entire skeleton.

However, the most pronounced degree of change and transformation is observed in the long tubular bones; then come the spine, sacrum, pelvis, skull, jaw bones and the flat bones of the thorax.[41] The short tubular bones, phalanges and tarsal bones, show the least transformation. When a long tubular bone is examined, one may find evidence of recent or healed fractures which may have produced gross bone deformity. The transverse diameter is apt to be increased at one or more points. The periosteum is usually thin. The medullary cavity may be filled with grayish-white fibrous tissue interspersed with fine reticulated bone. Single or multilocular cysts may be present, often extending to the periosteum. In and between such cysts, one may find evidence of recent or old hemorrhage. In other areas, the brown tumors of Von Recklinghausen (osteoclastoma) may be seen.

The vertebral column may be deformed, showing marked scoliosis or kyphosis. Because of the marked decalcification of the vertebral bodies, the elastic intervertebral discs expand, assuming an elliptical shape with resultant reduction of the superior-inferior diameter of many of the vertebrae, the fish-bone type vertebrae. Such changes account in part for the loss of height observed in the advanced cases.

Other gross skeletal changes may be present in the thoracic cage, the pelvis and cranium. The ribs are often atrophic, fractured, or irregularly shaped, thus producing a misshapen thoracic cage. The pelvis can become asymmetrical, misshapen and reduced in size. The bones of the skull may be atrophic and pliable; in other instances, sufficiently thickened to suggest Paget's Disease.[41] They can be cut easily with a knife. The increased fibrosis, cortical thinning, brown tumors or cysts may be demonstrable in any of the bony structures. The fractures usually occur at the site of a tumor or cyst.

On histological examination, one observes an increase of osteoclasts during any active phase of absorption, on the surfaces of haversian canals, trabeculae and Howship's lacunae. The tendency to resorption far exceeds new bone formation, which appears in the connective tissue. Thus increased osteoclastic activity is the special histological feature in hyperparathyroidism, the osteoblasts associated with new bone formation always being less pronounced than the osteoclasts. Both these cells are derived from the reticular cells of the bone marrow. The connective tissue increase and marrow fibrosis observed probably represents a proliferation of the reticular cells necessary for the formation of osteoclasts and osteoblasts. In such fibrous foci, giant cells and considerable extravasated blood may be present. The replacement of considerable bone marrow wih fibrous tissue will occasionally lead to sufficient decrease in the hemopoietic elements to cause an anemia and leukopenia. A bone may be exten-

*See footnote on previous page.

sively involved and yet show no brown tumors; if present, they vary in size and number. They appear as nests of cellular fibrous tissue containing large numbers of giant cells and blood pigment. These giant cells are identical with osteoclasts and therefore such cell nests are often called osteoclastoma. The brown color of such tumors is evidently dependent on hemorrhage. There is also considerable variability among the cysts. They may be entirely absent or very numerous. They may be microscopic in size or recognizable grossly. In the latter instance, if numerous, the bone gives the appearance of extensive cystic degeneration. They may be unilocular or multilocular, of sufficient size to expand the shaft of the bone and be the underlying cause of a fracture. Their possible mode of origin may vary. They may begin as a result of edema and degeneration of the fibrous tissue; such spaces may enlarge or fuse. Cysts may also result from degeneration occurring within the giant cell tumor areas.

From the above description, it is apparent that the skeletal changes of hyperparathyroidism are dependent on the activity of the hyperfunctioning parathyroid gland or glands as well as the duration of such hyperactivity. Other factors influencing the degree of skeletal change are the age of the patient, the calcium intake, pregnancies and the existence of other disease states. Thus a young adult who has always been on a high calcium intake might have hyperparathyroidism of short duration and yet show minimal or no bone changes on X-ray. If an elderly individual with osteoporosis develops the disease, the bone changes may be marked even though the degree of hyperparathyroidism is mild and of short duration. Therefore, in hyperparathyroidism the bone changes may not be demonstrable without microscopic examination, or one may encounter such marked bone changes (generalized osteitis fibrosa cystica) that the diagnosis is obvious on superficial inspection of the patient. It is probable that the degree of bone involvement is an index of the duration of the disease and not its severity.[33]

The hypercalcinuria and hyperphosphaturia of hyperparathyroidism are often responsible for the formation of renal calculi.[42] The formation of such calculi will depend on the amount of calcium and phosphorus excreted in the urine, the fluid intake, the alkalinity of the urine and the degree of polyuria. Thus the presenting symptom in an individual with severe hyperparathyroidism of short duration may be renal colic. In such instances the bones may be normal by X-ray. Thus hyperparathyroidism should be suspected in any patient with urinary stones.[33, 42] If this is done with regularity more patients will be so diagnosed before skeletal changes have occurred.

This and the other types of renal complications encountered in hyperparathyroidism have been studied in detail by Albright.[42] He divides the renal complications into three types:

1. Pyelonephritis secondary to the formation of calcium phosphate stones.

2. Nephrocalcinosis. In this type, calcium phosphate is precipitated out in the tubules, forming concretions which eventually lead to inflammatory changes, sclerosis and contracted kidneys. In certain instances the calcium deposits in the kidney (nephrocalcinosis) are demonstrable by X-rays. In such patients the hypophosphatemia may be absent, the hypercalcinuria decreased, the fecal calcium excretion increased and the partition of phosphorus in the urine as compared with the feces is less elevated. Patients with this type of renal lesion may be erroneously diagnosed chronic glomerular nephritis. In this connection, it should be pointed out that generalized hyperplasia of the parathyroids may occur as a result of chronic nephritis.[42]

3. Calcium deposits occurring in the kidney as well as other organs may take place in acute parathyroid poisoning with a resulting anuria and death in a few days. No other chronic renal changes should be present in this type of renal impairment. Metastatic calcification is an extremely rare finding post-mortem, but it can be produced with toxic doses of parathyroid extract.

Classification of Types of Hyperparathyroidism.—Therefore, depending upon whether the skeletal or renal pathology predominates and the degree of change present in either system, it is possible to describe several different types of hyperparathyroidism. Such a classification[33] of clinical types is given in the hope that every practitioner will appreciate that the other forms of hyperparathyroidism are not rarities but conditions which he will not infrequently meet.

1. Classical Hyperparathyroidism (Von Recklinghausen's disease or generalized osteitis fibrosa cystica). The skeletal signs and symptoms predominate and consist of pain, frequent fractures, bone

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deformities, cysts, tumors and generalized decalcification.

2. Osteoporotic Form of Hyperparathyroidism. This type is indistinguishable from the osteoporosis of osteomalacia, hyperthyroidism, inactivity or senile osteoporosis, except by means of the chemical or pathological laboratory. In such cases complete metabolic studies may be necessary to rule hyperparathyroidism in or out.

3. Hyperparathyroidism with Nephrolithiasis. The presnting symptom may be renal colic. Skeletal changes may be absent, minimal or maximal.

4. Acute Parathyroid Poisoning. There has been only one case reported which might have been so diagnosed and classified.[66]

5. Hyperparathyroidism Simulating or Complicated by Paget's Disease. Several patients have been reported as having both diseases. The exact relationship is unknown, but the author believes the existence of the two diseases in the same individual is coincidental.

Symptomatology and Physical Findings.—There is no one symptom which is diagnostic of hyperparathyroidism. Because of its insidious onset and extremely varied symptomatology, its existence is often not suspected and therefore often incorrectly diagnosed. Such patients are in consequence called hypochondriacal, neurasthenic, or incorrectely labeled rheumatism, arthritis, neuritis, bone tumor, flat feet, etc. Many of their complants being referable to the skeletal system, diagnostic X-rays may give the first clue, although they are also very often incorrectly interpreted as osteomalacia, osteoporosis, enchondroma, solitary cyst, renal rickets, sarcoma, etc. Many of the symptoms disappear immediately after operation on the parathyroids. Following operation, the patient is much more conscious of the previously existing vague symptoms.

Although no one symptom is characteristic of the disease, a thorough appreciation of the symptomatology will allow one to suspect it often and occasionally to make the correct diagnosis. The symptoms can be divided into three groups.[33] Those due to:

1. The hypercalcemia.
2. The skeletal changes.
3. The increased excretion of calcium and phosphorus.

Symptoms Due to Hypercalcemia.—The symptoms referable to the gastro-intestinal tract as well as those of a more general nature fall in this group. Those referable to the gastro-intestinal tract are anorexia, nausea, vomiting, pain and constipation. Anorexia and constipation are common and may be the presenting symptoms. Intractable nausea and vomiting are occasionally present. Lassitude, weakness, easy fatigability and weight loss may be the only symptoms early in the disease. Hypotonia is common and probably is the cause of some of the skeletal symptoms. This may involve the joint structures as well as the muscles. The hyperextensibility and hyperflexibility may be sufficiently great to allow the patient to do contortionist tricks. The hypercalcemia also causes decreased excitability of the nerves, but such determinations are neither practical nor necessary.

Symptoms Due to Skeletal Involvement.—From what has been said before, it must be apparent that the symptoms falling in this group are dependent on the severity of the case, its duration, etc. Therefore, one may encounter patients with no bone symptoms; in others they represent the major complaint, such as spontaneous fractures, bone pain, bone tenderness, bone tumor, kyphosis, loss of height, waddling gait or limp. The tumors of the jaw are often called epulis. Bone deformities are a later manifestation with the exception of the spine.

Symptoms Related to the Hypercalcinuria and Hyperphosphaturia.—Polyuria and polydipsia are usually present and may be so marked as to suggest diabetes insipidus. The large quantities of water necessary for the increased excretion of calcium and phosphorus are responsible for the polyuria. This fluid loss is compensated for by an increased thirst and subsequent increased fluid intake. Nocturia may be a distressing feature of the polyuria. Enuresis is occasionally present. Painful urination may be caused by the passage of gravel or a small stone. Renal colic or some other manifestation of urinary calculi may be the first and only symptom. In such instances, no bone changes may be demonstrable by X-ray. Ten patients, in a series of twenty-five proven cases, were discovered at the Massachusetts General Hospital as a result of doing routine serum calcium and phosphorus determinations on all patients with urinary calculi. Only five of the twenty-five cases had bone disease without renal disease. Therefore, we are of the belief that the nephrolithiasis type of hyperparathyroidism may in the future be the commonest type to be recognized.

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X-rays.—The roentgenologist will have no difficulty in making the diagnosis, with marked generalized osteoporosis, cysts, tumors, fractures, bone deformities and a granular, mottled appearance of the skull.[43, 44] However, one must remember that Von Recklinghausen's disease is rare and not representative of the commoner types of hyperparathyroidism. As previously stated, skeletal changes may not be demonstrable by X-rays. The presence of cysts, bone tumors, fractures or deformities is not sufficient for an X-ray diagnosis. Being a metabolic disease, hyperparathyroidism must exert its fundamental action, demineralization, on the entire skeleton. Therefore, if generalized decalcification is not associated with such cysts, tumors, etc., one is forced to conclude that he is dealing with a localized disease. From the pathologic description it is obvious that X-ray findings are not pathognomonic except in the classical form. The bones may show no demonstrable change. Again, a generalized decalcification indistinguishable from that observed in osteomalacia, senile osteoporosis, multiple myeloma, or pituitary basophilism may be the only demonstrable deviation from the normal. In such instances, lateral roentgenograms may disclose the biconcave vertebral bodies (fish-bone vertebrae). They are merely an expression of the bone softening present and are seen in other diseases with associated generalized decalcification. Cysts may be entirely absent, numerous, vary considerably in size, or be present in only one or two bones such as the metacarpals. There may be expansion of the overlying bone. In long-standing cases, there is marked loss of substance in the terminal phalanges. The loss of supporting structure results in clubbing of the fingers. Tumors may occur in any portion of the skeleton but are more common at the ends of the long bones or in the ribs and jaws. Absence of the lamina dura may suggest the diagnosis but is only a manifestation of generalized decalcification.[45] The miliary mottled and granular appearance is most commonly observed in the flat bones, particularly the skull. This appearance is due to newly formed, closely set trabeculae which have replaced the original bones. However, the roentgen appearance of the skull is variable; in some instances it is thickened, the thickening being generalized or irregular.

Therefore, unless advanced bone resorption and numerous newly formed trabeculae are present, the granular, mottled appearance is not demonstrable. In long-standing cases, the long bones may be practically free of trabeculae, the cortex extremely thin, and the shadow cast gives a very light, homogeneous appearance. In such cases, the bone cysts, tumors, fractures, bone deformities, etc., will probably be present. In a rare case, generalized decalcification may be associated with a lesion or lesions characteristic of Paget's disease. Whether this is anything more than coincidence cannot be definitely stated at this time. Thus it is obvious that X-rays taken for other reasons may give the first clue of the existence of the disease.

Differential Diagnosis.—From the above, it must be apparent that hyperparathyroidism is a distinct disease entity,[46] which may mimic several other diseases. Conversely, many diseases may mimic hyperparathyroidism. Therefore, one must appreciate the other causes of the varied symptomatology observed in hyperparathyroidism. It is easy to understand why other bone diseases might be misdiagnosed hyperparathyroidism. Once the diagnosis is suspected, its confirmation or exclusion is dependent on the chemical or pathological laboratory. In most instances determination of the calcium and phosphorus content of the serum suffices, occasionally complete metabolism studies are necessary, rarely a bone biopsy. In the mild border-line case (the osteoporotic type), one may be required to resort to all available diagnostic measures. In very rare instances, such observations may have to be repeated at some subsequent date in order to rule hyperparathyroidism in or out. Early confirmation or exclusion of hyperparathyroidism is necessary if we are to prevent the marked skeletal changes and complications of long-standing hyperparathyroidism from occurring. Certainly one is not justified in advising exploration of the parathyroid glands because of the existence of demonstrable skeletal changes until he has obtained confirmatory evidence from the chemical or the pathological laboratory that such skeletal changes are due to hyperactivity of the parathyroid glands. For this and other reasons we cannot sanction parathyroidectomy in patients suffering from arthritis,[46, 47, 48] osteitis deformans[49, 50, 51] and other unrelated diseases,[52] as advocated by certain workers. The skeletal diseases most often confused with hyperparathyroidism which must therefore be excluded are:

Senile Osteoporosis.—The term, osteoporosis, implies rarefaction of the skeleton. Such bones are

very transparent to roentgen rays. Its presence is of no diagnostic significance. It occurs in all aging and senile persons; therefore, the term, senile osteoporosis. It must not be confused with the osteoporosis resulting from inactivity, an inadequate diet or endocrine diseases (such as hyperparathyroidism, hyperthyroidism, or a basophilic pituitary adenoma). The conditions operating to produce senile osteoporosis are those related to the production of the general involutionary process of aging. According to Pommer,[53] in senile persons the physiological deposition of new bone is decreased, whereas physiological resorption continues; in consequence, there results a progressive thinning of the bone. Therefore, one observes the kyphosis of the aged, the fish-bone type of vertebrae and fractures, particularly of the neck of the femur. Grossly, one observes fatty atrophy of the marrow, thinning of the cortex and trabeculae. Microscopically, the osteoid borders are not demonstrable and there is no increase of osteoclasts, marrow fibrosis, etc., as seen in hyperparathyroidism.

X-rays of such patients may show such marked rarefaction as to suggest hyperparathyroidism. It resembles the mild osteoporotic form with borderline blood values and therefore may be difficult to differentiate. The symptoms of hyperparathyroidism due to the hypercalcemia and hypercalcinuria are absent, the skeletal symptoms minimal. Bone pain may be present; fractures and deformities may occur. The chemical findings usually enable one to differentiate it from hyperparathyroidism. The serum calcium is normal, the serum phosphorus normal or reduced, and the serum phosphatase normal. If such data are not sufficient to exclude hyperparathyroidism, metabolic studies and bone biopsy may be necessary. In such instances the bone biopsy is of diagnostic importance.

Osteomalacia.—Osteomalacia has often been confused with osteitis fibrosa. Particularly was this true prior to our better understanding of calcium and phosphorus metabolism in health and disease. This was due in part to the fact that parathyroid hyperplasia is sometimes present in osteomalacia.[54] However, Erdheim, as early as 1907, postulated that such hyperplasia was the result of the bone disease and not the cause of it (compensatory hyperplasia). Since then it has been definitely established that osteomalacia is a calcium and phosphorus deficiency disease resulting from a low calcium intake and vitamin D deficiency. The state of pregnancy and lactation will accentuate the disease. It does occur endemically in certain regions such as northern China, India and Japan. In this country it is a rare disease, its incidence being lower than that of hyperparathyroidism. Most of the cases we have observed have been individuals with a fatty diarrhea and a resulting lack of absorption of the fat-soluble vitamin D.

Because of the existing vitamin D deficiency and lack of absorption of adequate amounts of calcium and phosphorus, the bone tissue shows a definite pathological abnormality, namely, a failure of calcium and phosphorus deposition in the osteoid tissue. In consequence, one observes wide osteoid borders. Osteoblasts are far in excess of osteoclasts. There exists a less marked increase of fibrous tissue, etc., than is seen in osteitis fibrosa. Here, again, a bone biopsy is pathognomonic.

The symptoms complained of are few as compared with hyperparathyroidism. Although skeletal pain is common, the presenting symptom may be secondary to the collapse of a decalcified fish-bone vertebrae. In some patients the symptoms due to the associated infantile type of tetany are the reason for seeking medical aid. The bones bend rather than fracture. The roentgenograms show generalized decalcification. The serum calcium is normal or low, the serum phosphorus is low and the serum phosphatase high. In such cases, an adequate calcium and phosphorus intake and sufficient vitamin D results in a rapid therapeutic response.[9, 55]

Osteoporosis of Hyperthyroidism.—This form of generalized osteoporosis is sometimes seen in long-standing exophthalmic goitre. The osteoporosis of hyperthyroidism is evidently due to increased lacunar absorption. It may persist for years after. It has been previously discussed under normal calcium and phosphorus metabolism.

Osteoporosis of Disuse or Inactivity.—Generalized osteoporosis of this type is occasionally encountered in bed-ridden patients with reduced activity such as ankylosing arthritis. Here the cause of the decalcification should be obvious. The blood chemistry is normal.

Osteoporosis Due to Basophilic Adenoma of the Pituitary.—Generalized osteoporosis[56] is also observed in this recently described syndrome (Cushing's syndrome).[57] There is some evidence to suggest that this type of osteoporosis may be due to

hyperplasia of the parathyroid glands. In one of his cases, calcium and phoshorus metabolism studies revealed a normal serum calcium, a reduced serum phosphorus and a hypercalcinuria. The bone biopsy, however, more nearly resembled that seen in senile osteoporosis. Whether this is or is not a distinct disease entity, the existing obesity, hirsutism, purplish atrophic striae, amenorrhea and hypertension should serve to differentiate it from the osteoporotic type of hyperparathyroidism.

Osteoporosis Due to Suprarenocortical Tumors.—A syndrome presenting many of the characteristics of Cushing's syndrome is occasionally due to a suprarenal adenoma.[58] Surgical removal of the adenoma in several instances has been followed by complete regression of the abnormalities characterizing the syndrome. Autopsy in two other cases[58] revealed a normal hypophysis. Osteoporosis, although present, was confined chiefly to the vertebral column. The blood values were similar to those described under Cushing's syndrome. Here, again, the physical characteristics of the syndrome prevent one from confusing it with hyperparathyroidism.

Paget's Disease.—Paget's disease and hyperparathyroidism have very little in common. The signs and symptoms of the two diseases are not the same. There is no evidence to substantiate the view held by some that Paget's disease is a form of hyperparathyroidism.[49, 50, 51] The reasons for contradicting such a view are:

1. In Paget's disease, the changes are sometimes confined to one lone bone and, as Schmorl[59] points out, although often polyostatic, it is never generalized. However, in hyperparathyroidism, as one would expect in a disease of endocrine origin, the effect is a generalized one, every bone in the body being involved. This may not be apparent in the gross but is on microscopic examination.

2. Paget's disease occurs in older people, usually males, and is regularly associated with arteriosclerosis, whereas hyperparathyroidism is met with in all age groups, more commonly in females, and arteriosclerosis is not a common finding.

3. Schmorl[59] was unable to show any significant histological changes in the parathyroid glands of 138 autopsied cases of osteitis deformans.

4. The calcium and phosphorus metabolism changes are not of the type observed in hyperparathyroidism. The serum calcium is normal or only slightly elevated and the serum phosphorus slightly elevated, normal, or slightly reduced. The serum phosphatase is always elevated, being much higher in a case of Paget's disease than in one of hyperparathyroidism with a comparable degree of bone involvement. In a few cases we have observed slight increases in the calcium and phosphorus excretions, but the increased excretions were not confined solely to the urine as they are in hyperparathyroidism.

There are other differences. Malignant bone tumors develop in a fair number of cases of Paget's disease; such is rarely, if ever, observed in hyperparathyroidism. Fractures occur very often in hyperparathyroidism and seldom in Paget's disease. Renal calculi do occur but are less frequent than in hyperparathyroidism.

It may be difficult or occasionally impossible to distinguish histologically Paget's disease from hyperparathyroidism. However, in Paget's disease new bone formation is always in excess of bone resorption. The newly formed bone of Paget's disease takes on a distinguishing mosaic appearance because of the numerous thick, irregular, dark staining cement lines.

Roentgenograms usually serve to separate the two diseases. In Paget's disease, lesions may be confined to one or two bones. In most cases one can demonstrate some normal bone structure, most frequently in the small bones of the hand. The bones involved may be bowed and show the characteristic cortical enlargement (hyperostosis) as compared with the normal or thin cortex of osteitis fibrosa. The moth-eaten appearing skull usually shows this same thickening as compared with the thin moth-eaten skull of most patients with hyperparathyroidism. As Schmorl[59] pointed out, the bones most frequently involved are those subjected to the greatest stress and strain.

If all of these facts are taken into consideration, there is very little excuse for confusing the two diseases or thinking they are causally related. Nevertheless, we have included, under clinical types,[33] "hyperparathyroidism simulating or complicated by Paget's disease." In the two cases observed in this clinic, the roentgenograms were characteristic of hyperparathyroidism except for typical focal lesions of Paget's disease. In one case, the existence of both diseases has been proven by bone biopsies. These isolated cases may represent the coincidental

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finding of both diseases in one individual. However, the hyperparathyroidism with its resulting increased osteoclastic activity may have been responsible for making manifest a focal lesion of Paget's disease which would have otherwise remained dormant. Routine calcium and phosphorus determinations on all patients with Paget's disease may disclose more cases of this sort, although this has not been our experience in a large series of cases of Paget's disease.

Renal Rickets.—There seems little doubt but what chronic renal insufficiency may lead to rachitic-like changes in the skeleton.[60] These bone changes have been attributed to the existing acidosis. In one instance the daily administration of large doses of soda bicarbonate resulted in correcting the acidosis and healing of the bone lesions.[61] In all cases of so-called renal rickets, it is important to establish whether one is dealing with true renal rickets or hyperparathyroidism, with associated renal lesions described as the type II lesions.[42] The evolution of the symptomatology, bone symptoms antedating the renal symptoms, or *vice versa,* will often be of assistance. In either instance, the serum phosphorus is elevated. In true renal rickets the calcium is depressed, whereas, in hyperparathyroidism with type II renal lesions the serum calcium is normal or elevated.

Solitary Cysts.—Single cysts usually occur in the epiphyses of the long bones. They may occur in more than one bone. Histologically, they are indistinguishable from the cysts observed in hyperparathyroidism. Such cases can be distinguished from hyperparathyroidism by the demonstration of an otherwise normal skeleton. The serum calcium, phosphorus and phosphatase are all normal. They may represent a healing process secondary to trauma or infection. Some probably represent healing reaction in a giant cell tumor.

Solitary Benign Giant Cell Tumor.—These tumors may resemble histologically the giant cell tumors of osteitis fibrosa. Here again, the blood values are normal and generalized osteoporosis is not demonstrable.

Osteogenesis Imperfecta (Fragilitas Ossium, Brittle Bone).—Osteogenesis imperfecta might be confused with osteitis fibrosa because of thin bones and frequent fractures. However, such confusion is hardly justified in the presence of blue sclerae and deafness. It is a hereditary disease, characterized by failure to form normal osteoid tissue. The blood chemistry is normal. The calcium and phosphorus excretions are not increased.

Multiple Myeloma.—Multiple myeloma may simulate hyperparathyroidism. Decalcification, with or without mottling, fish-bone type of vertebrae (often crushed), irregular areas of decreased density and areas of bone destruction are often demonstrable. The decalcification is rarely if ever generalized. Hypercalcemia is often present. It fluctuates considerably more than in hyperparathyroidism and may or may not be associated with an increase in serum proteins. The serum phosphorus is usually elevated; rarely does it approach the hypophosphatemia level characteristic of osteitis fibrosa. The serum phosphatase is normal. Secondary renal changes are frequent in multiple myeloma and compensatory parathyroid hyperplasia has been observed. Bence-Jones protein, if present, does not establish the diagnosis of multiple myeloma because it can occur in Von Recklinghausen's disease. However, in such cases the Bence-Jones proteinuria is minimal, whereas, in multiple myeloma it is present in a marked degree. In occasional cases bone biopsy may be necessary to establish the correct diagnosis.

Metastatic Malignancy.—The differentiation of this condition from hyperparathyroidism should never cause serious difficulty. Generalized decalcification is not present, the uninvolved bones are normal. Metastases seldom occur below the knees or elbows. Hypercalcemia has been reported but without a co-existing hypophosphatemia.

Lipoid Dystrophies:

(a) Schuller-Christian's Disease. — Schuller-Christian's disease should not be confused with hyperparathyroidism. It occurs most frequently in early childhood. Roentgenographically, the bone changes represent a destructive process, usually of the flat bones. The unaffected bone or uninvolved portions of the same bone exhibit a normal structure in contrast with the diffuse involvement of hyperparathyroidism. The blood chemistry in the few cases observed has been normal.

(b) Gaucher's Disease.—Gaucher's disease may show some generalized decalcification secondary to bone marrow invasion. Such decalcification is more apparent in the ends of the femur and humerus. Skull changes appear very late in the disease. The

anemia, splenomegaly, etc., enable one to differentiate it from osteitis fibrosa.

Erythroblastic Anemia.—Although the roentgenograms may simulate hyperparathyroidism, clinically, the differentiation is not difficult.

Radium Poisoning.—Here, again, the sharply demarcated areas of bone destruction without generalized decalcification are sufficient to rule out hyperparathyroidism.

Treatment:

Medical.—This falls under two headings, roentgen therapy and dietary treatment. No beneficial metabolic or clinical results have been observed in this clinic in treating hyperparathyroidism with roentgen rays. The cases so treated thus far have all been due to parathyroid adenomas. Whether one would experience the same results in cases due to generalized hyperplasia is unknown. Unfortunately, we have no way of differentiating these two types of hyperparathyroidism without exploration of the parathyroid glands. One can prevent decalcification as well as demonstrate skeletal improvement with a high calcium, a high phosphorus, or a high calcium-high phosphorus diet. However, such treatment increases the danger of renal complications and is therefore contraindicated.

Surgical.—The surgical treatment of hyperparathyroidism has been very thoroughly discussed by Churchill, Cope, and Walton.[63, 64, 65] It is unusual to be able to demonstrate a palpable parathyroid gland on physical examination. This is due to the fact that they lie deep in the neck or mediastinum, as well as the ability of soft parathyroid tissue to mold itself to conform to the surrounding structures.

Having established a diagnosis of hyperparathyroidism, there is no way of determining prior to operation whether it is due to a lone adenoma or hyperplasia of all parathyroid glands. In the case of a large parathyroid tumor, the roentgenologist may be able to locate the tumor. Therefore, such pre-operative studies should be made in all cases. X-rays of the upper mediastinum may demonstrate a calcified encapsulated tumor or a displaced trachea. A substernal goitre may occasionally be misleading. The tumor may lie in close proximity to the esophagus. Therefore, anterior and lateral fluoroscopy during the administration of thick barium may be of help.

The chief operative difficulty is locating the parathyroid tumor. The surgeon must know the normal and aberrant positions of the parathyroid glands. He must realize that they may be encountered anywhere from the pharynx to the mediastinum, although none have been found above the upper poles of the thyroid. They are often in contact with the upper or lower poles of the thyroid. However, they have been located in the anterior and posterior mediastinum, behind the esophagus and trachea and in the thyroid sulcus. Parathyroid tumors have been recorded as buried in the thyroid gland.

The first surgical exploration of the neck offers the best chance of finding the parathyroid adenoma. The scar tissue of a previous operation greatly increases the difficulties. Meticulous hemostasis is essential, as staining of tissue with blood may make the recognition of parathyroid tissue impossible. Therefore, Churchill advises the ligation of even the smallest vessels before sectioning, the most careful handling of all tissues and sharp dissection, because the slightest trauma to a parathyroid gland and the resulting hematoma may completely mask its identity. Furthermore, such trauma or interference with the vascular supply of the normal glands enhances the possibility of post-operative tetany.

Parathyroid glands have a characteristic reddish brown color and smooth surface. They are most often confused with lymph or hemolymph nodes, lobules of thyroid tissue and fetal fat. With experience the parathyroid glands can be identified with great accuracy, because of their shape, color and tiny vascular pedicle. Given a marked degree of hyperparathyroidism one can expect to find a sizable tumor. The smallest gland encountered in this clinic was one measuring 1x0.5x0.5 cm. The patient had the osteoporotic type of hyperparathyroidism of minimal degree. A small amount of normal parathyroid tissue was still demonstrable at the periphery of the gland. Therefore, one need not stop to examine each minute body of tissue encountered. Furthermore, when operating on a patient with an unequivocal diagnosis of hyperparathyroidism, the removal of normal parathyroid tissue is absolutely contraindicated because no beneficial results will ensue and the danger of post-operative tetany is greatly enhanced when the tumor is removed at a subsequent operation. The removal of a lobe of the thyroid is not justified until a careful block dissection of the neck and palpation of the

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mediastinum have been made. If no tumor is found after such thorough exploration, the diagnosis must be rechecked by repeating the metabolic studies and performing a bone biopsy. The latter can be made at the conclusion of the first exploration. If all data are still consistent with the diagnosis of hyperparathyroidism, exploration of the mediastinum by section of the sternum is indicated. In the one case in which this operation was performed, the tumor was found lying just below the left innominate vein. The patient had had six previous operations in an attempt to find the tumor. Thus, with some knowledge of the possible locations of the parathyroids and patience, the surgeon will usually be able to identify all of the parathyroid glands. Such an extensive search is not indicated unless one is dealing with hyperparathyroidism due to generalized hyperplasia and not an adenoma.

In order to establish which type of hyperparathyroidism one is dealing with as well as the extent of the neck exploration necessary, the surgeon should have a pathologist present at the time of the operation. He can determine by means of a frozen section from a biopsy specimen whether he has disclosed parathyroid tissue. If it is, he can further tell whether it is normal, hyperplastic or representative of an adenoma. If normal, one can exclude osteitis fibrosa due to general hyperplasia of the parathyroids. If it is hyperplastic, the surgeon should continue his search until all parathyroid glands have been identified. If an adenoma is disclosed, one can usually assume it represents the causative agent, although occasionally two exist.

In the patient with adenoma of one parathyroid gland, one is tempted to remove it *in toto*. In this clinic such is done providing one or more normal parathyroid glands have not been removed at some previous operation. If such is the case, a subtotal resection is done in order not to run the risk of producing the serious complication of permanent hypoparathyroidism. The post-operative tetany following the removal of a parathyroid tumor may be dangerous. The most severe tetany we have observed has occurred in patients with the most extensive decalcifiation. In one such instance where the remaining parathyroids were presumably intact and had not been disturbed at operation, very serious tetany ensued following the removal of the parathyroid tumor. In such cases we believe the tetany results because of too rapid deposition of calcium and phosphorus in the previously depleted bones and not because of atrophy of disuse of the other parathyroid glands. Therefore, in such cases one should never attempt more than a subtotal resection of the tumor. If this is done, severe tetany of months' duration will be avoided. If the surgeon chooses to do a subtotal resection, he should leave behind, with a good blood supply, a piece of tumor considerably larger than a normal gland. Its location should be clearly marked by a silver wire suture. If there is a recurrence of the hyperparathyroidism, removal of the remainder of the gland should offer no difficulty.

The treatment of generalized hyperplasia of all parathyroids cannot be definitely stated at this time. It is obvious that total surgical removal is contraindicated. The surgeon must make every effort to see all parathyroid tissue in order to know exactly what is being removed and what is being left behind. To date, it has been the practice in this clinic to remove approximately three-quarters of the total parathyroid tissue identified.[35, 64] Following this simple rule has given satisfactory results to date in the three cases so treated in this clinic. It is better to err on the safe side than to induce a chronic parathyroprivic state. Residual tissue with a good blood supply left in position some distance from the recurrent laryngeal nerve will be readily accessible if a secondary operation is indicated.

Post-Operative Course.—Following parathyroidectomy for either type of hyperparathyroidism, metabolic changes are demonstrable in a few hours. The serum calcium falls extremely rapidly, as much as 4 to 5 mgm. in twelve hours. The phosphorus may fall slightly and is slow to rise to its normal value. The plasma phosphatase if elevated may take weeks or months to return to its usual normal level. A marked decrease in the excretion of calcium and phosphorus occurs very promptly (hours). Some patients have become practically anuric. The urinary excretion of calcium and phosphorus may occasionally remain subnormal for a period of months. Tetany may develop at as high a serum calcium level as 8 mgm. per 100 c.c. Any of the previosuly mentioned symptoms may appear. The treatment indicated has been fully discussed under hypoparathyroidism.

Following operation, the improvement in sympto-

matology is soon noted and at times is most dramatic. The bone pain may be one of the first symptoms to disappear despite the fact that increased radiability of the bones may be present for months. Recalcification of the skeleton in certain instances has required months. The bone tumors (osteoclastoma) gradually disappear, the bone cysts remaining. Thus, post-operatively, if a previously described lesion disappears, one can infer that it was a tumor, whereas, if it persists, it is likely that it was a cyst. I doubt if the calcium deposits in the kidney (nephrocalcinosis) are ever reabsorbed. The improvement in general health is so marked that patients frequently state that they have not felt so well in years. Thus, disappearance of all symptoms, whether referable to the skeleton, the hypercalcemia, or the increased excretions of calcium and phosphorus is to be noted and, as a general rule, very promptly. Naturally in the classical cases of Von Recklinghausen's disease, the marked skeletal deformities persist.

SUMMARY

Hyperparathyroidism is a distinct disease entity due either to adenoma of one or more of the parathyroid glands or to generalized hyperplasia of all the parathyroids. Although its clinical manifestations are varied, the calcium and phosphorus metabolism changes are characteristic. Following removal of the responsible hyperfunctioning parathyroid tissue, clinical improvement is most striking. The disease should be recognized sufficiently early to allow for complete restitution of the patient. If the disease is not arrested, it continues to progress, and the patient succumbs to one of the complications of a disease, the treatment for which is highly successful.

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VOMITING IN THE NEWBORN, WITH SPECIAL REFERENCE TO DUODENAL ATRESIA—REPORT OF TWO CASES.*

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Vomiting in the newborn is from either functional or organic causes. Functional vomiting is far more frequent, but often not much thought is given to the possibility of an organic cause until the child is exhausted from starvation and dehydration. In the young infant, the first vomiting should bring to mind the thought that the vomiting is the result of either a functional disturbance or an organic lesion and that an early differentiation of these causes is of extreme importance. If this were done, a diagnosis would more frequently be made before the baby becomes a poor operative risk or is in extremis.

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In functional vomiting the child does not appear sick. Vomiting may be the result of too large a quantity of milk at each feeding causing an overflow, or it may be caused by too frequent feedings. The most common cause of functional vomiting is an autonomic imbalance or spastic child. This condition can readily be controlled by atropine in a one to one-thousand solution given in one drop doses ten minutes before each feeding and increased one drop at each dose until the vomiting is controlled or the physiological effect of the drug obtained.

Vomiting caused by organic lesions presents a different picture. The child usually looks sick, is uncomfortable, the skin is wrinkled and dry, and

the face is pinched because of the loss of fluid. It has been said that the body can lose one-half of its proteins and still live, but if it loses one-tenth of its water it will die. So the infant shows very quickly the effect of the loss of water. Vomiting caused by an organic lesion is projectile in type and at times explosive. If the infant is watched after feeding, often it will be observed that the baby becomes restless, squirms about and then with an explosive suddenness vomits the whole feeding. Constipation is present, though here it is easy to be deceived. If the chart or the nurse's word is relied upon one will often be misled and both may report several stools a day without any reference to their character. The frequent bowel movements are often meconium stools and contain no milk. Weight loss is constant and usually rapid. Therefore, by contrasting the appearance of the child, the type of vomiting, the rapid loss of weight and the character of the stools, it may be determined whether the vomiting is functional or organic.

After the conclusion is reached that an organic lesion is responsible for the vomiting, it then becomes necessary to determine the kind of lesion that is responsible for the condition. A cerebral hemorrhage may cause vomiting, but the type of labor, the refusal of the child to nurse, the increased intra-cranial pressure and lumbar puncture will make the diagnosis. Imperforate anus or stricture of the rectum should be considered. A digital examination is all that is necessary to clear this up. The most frequent organic lesion that causes vomiting is congenital pyloric stenosis. It is necessary that this condition be differentiated from an atresia of the bowel, the most frequent site of which is in the duodenum. In both conditions, vomiting is similar in type, projectile, the weight loss is rapid and constipation is present. However, there are a few cardinal symptoms that will be helpful in differentiating the two.

In congenital pyloric stenosis vomiting comes on usually a week or two after birth. In atresia, the vomiting begins immediately, or within a day or two after birth. In pyloric stenosis, peristaltic waves are visible in the epigastrium. After a feeding, peristaltic waves can be seen to start at the pylorus and pass upward, diagonally, across the epigastrium toward the cardia. In atresia, intestinal peristalsis is visible below the epigastrium, and at times the waves have a step-ladder appearance. An X-ray examination is helpful in differentiating the two conditions. In stenosis the contrast medium, barium, is shown to remain in the stomach. In atresia the intestine is distended with gas above the point of obstruction. If the obstruction is not complete gas pockets may be seen scattered over the abdomen. Barium will then show the point of greatest obstruction. However, it is usually best not to give barium if atresia is suspected as it may increase the obstruction.

Dr. Sidney Farber, pathologist to the Children's Hospital at Harvard University, has worked out an ingenious test for atresia of the bowel. The test is dependent upon the embryological development of the child (Ladd, Wm. E.: Congenital Obstruction of the Small Intestine, *J. A. M. A.*, 101:1453-1457, November 4, 1934). "Prior to the fifth week of fetal life the intestine presents a well defined, round lumen lined with epithelium. Soon after this the epithelium rapidly proliferates, and the lumen of the intestine becomes obliterated by epithelial concrescence. The solid stage persists for a short time until vacuoles appear and coalesce to re-establish permanently, by the twelfth week of fetal life, the intestinal lumen. An arrest in development during this period results * * * in atresia of the intestine at one or more points * * * ."

Meconium begins to collect in the intestines after the twelfth week of fetal life. Meconium is made up of mucus, bile, keratinized epithelial cells, lanugo hairs and vernix caseosa swallowed with the amniotic fluid. Vernix caseosa and keratinized epithelial cells are not present in large quantities in the amniotic fluid until the last few months of pregnancy. Of these elements, keratinized epithelial cells are the easiest to detect. They may be detected in a "smear dried with ether, stained with Sterling's gentian violet for one minute and decolorized with acid alcohol. Failure to find these keratinized epithelial cells in the stools is an indication that one is dealing with a case of atresia."

Case 1.—A baby girl, born six weeks premature, vomited all food and water from birth. She was treated by one of us (Falkener). The patient showed symptoms of obstruction of the duodenum, which was confirmed by X-ray examination. The patient was operated upon (by Horsley) on September 20, 1932, at the age of six days. The opera-

tion was a posterior gastro-enterostomy. The child's condition was quite desperate. The operation was done with local anesthesia and a sugar rag. Ringer's solution had been given by hypodermoclysis before operation. After the operation more Ringer's solution was given by hypodermoclysis and by proctoclysis. The baby died of shock about four hours after the operation. Post-mortem examination showed that the pylorus was greately dilated, as was the proximal duodenum. On further exploration the duodenum was seen to end in a cul-de-sac with no opening in its distal end. The jejunum began in a pouch, and there was no connection between the termination of the duodenum and the beginning of the jejunum.

Case 2.—The baby was a boy, born May 1, 1934, and weighed 5 pounds and 6 ounces at birth. Seven and one-half hours after birth the baby was put to breast, but refused to nurse. He vomited a large amount of dark brown fluid. He was treated by one of us (Walker). At 10 P. M. the baby nursed fairly well, but vomited the 2 A. M. nursing. The vomiting continued, projectile in type. On the second day artopine 1:1000 in one drop doses was given, and increased to five drops without results. There were two to four small mucous stools daily. On the fourth day an X-ray examination showed small gas pockets over the abdomen. He was operated upon (by Horsley) at the age of five days. The condition appeared similar to that in the first case, only not quite so far advanced. The pylorus and upper duodenum were widely dilated and there was an obstruction in the duodenum rather low down because the child would vomit bile. On account of the condition of the baby no extensive exploration was made. A gastro-enterostomy was done under ether anesthesia. After the operation salt solution was given by hypodermoclysis, and 1 per cent dextrose in normal saline solution was given by proctoclysis. The patient made a satisfactory recovery. He is now, October, 1934, five months old, is doing well, and weighs 14 pounds and 2 ounces.

COMMENT

Congenital duodenal atresia has not received the attention it deserves. The diagnosis has been discussed, and the importance of differentiating as soon as possible functional early vomiting of an infant from vomiting caused by an organic lesion has been emphasized. It seems highly probable that in the State of Virginia many of these infants that might be saved by prompt diagnosis and proper treatment are perishing. If in the experience of ourselves (two pediatricians and one surgeon), there have occurred within a period of two years two cases of congenital duodenal atresia, there must be many more cases of this kind scattered throughout the country.

Too frequently is a diagnosis made either of a functional condition or of improper feeding, and different feeding formulae are tried and various drugs administered until the baby dies. Then the death is ascribed to some form of asthenia or a similar vague disturbance. A necropsy is seldom done. Babies as well as adults usually die of very real things, and rarely of functional disturbances. If physicians who attend babies will bear in mind the gravity of the symptom of early vomiting, and will not be content until the cause of the vomiting has been definitely ascertained and proper treatment instituted, many babies that now die within the first few days of their lives, with vomiting as a chief symptom, can be saved.

TREATMENT

The treatment of congenital duodenal atresia is obviously surgical. The operation should usually be a gastro-enterostomy.

A gastro-enterostomy in these infants is quite different from that done in an adult. It must be performed on an infant under ten days or two weeks old, because in complete duodenal atresia the little patients would probably not live longer, even with hypodermoclysis and the rectal administration of salines and water. The jejunum is small and contracted, no larger than a quill. The transverse mesocolon is veil-like and transparent. It is not necessary to incise the transverse mesocolon, but it may be included in the suture.

Fine silk in a small curved needle is the best suture material for these cases. Two tractor sutures are placed between the loop of jejunum that it is proposed to unite to the stomach, and the posterior wall of the stomach, and are clamped and held taut. In very young infants the contents of the stomach and upper small intestines are practically sterile, but a small moist gauze pack should be placed around the site of operation.

On account of the small size of the intestines, too

much suturing is undesirable, so, instead of inserting a preliminary continuous suture between the jejunum and stomach before incising the stomach and jejunum, the incision is first made, and a row of silk sutures placed as a continuous lockstitch and drawn snugly is the only suture used posteriorly. This line of sutures, accurately placed, is safe. When it is brought anteriorly, however, there is some difficulty in making as smooth a union as in the posterior wound, so an additional row of sutures is inserted anteriorly, and this may be reinforced by a few interrupted mattress sutures of fine silk.

Because of the active peristalsis of the stomach and intestines, and the possibility of rapid absorption of catgut, together with the fact that the danger of the use of silk in the stomach is generally much over-rated, silk or linen is always used in these cases.

It is highly important that there be no obstruction to the afferent or efferent loop. The lumen of the bowel is so small that an obstruction could easily occur. Using only one row of sutures posteriorly lessens such a possibility. (Horsley, J. Shelton: Surgery of the Stomach and Duodenum, C. V. Mosby Co., St. Louis, 1931).

CONCLUSION

Congenital duodenal atresia is probably far more common than it appears to be. Doubtless many cases of early vomiting in infants are from this cause, and unless the condition is recognized in the first few days of life and operation done promptly, death is inevitable. The recognition of this state of affairs by pediatricians and general practitioners who treat infants will greatly aid in lowering the mortality of early infancy.

DISCUSSION

DR. J. SHELTON HORSLEY, Richmond: I am glad to have the experience of being associated, as co-author, with these two gentlemen. Our experience with atresia is interesting. As Dr. Walker says, if three of us in two years have found two cases of congenital duodenal atresia, it is extremely probable that there is a great deal of it unrecognized. As he says, these babies are treated for vague disorders; they die, and usually no necropsy is made, and the death is put down as from asthenia or something else. Doubtless in the State of Virginia there are many, many babies dying of this same condition that is not recognized.

In regard to the technical part of the operation, it is quite different from that in an adult. It is described in the paper.

I want to commend the splendid work in the diagnosis of these two cases by Dr. Walker and Dr. Falkener. Both cases came to me diagnosed as congenital duodenal atresia, and they proved to be just that. Dr. Walker has given us the findings on which the diagnosis was made, and the first symptoms. A surgical operation, usually gastro-eneterostomy, is the only treatment.

DR. WALKER, closing the discussion: We should keep in mind the necessity of making a differential diagnosis when the newborn baby vomits persistently. We are inclined to think that vomiting in the newborn baby is a simple matter. Usually it is, but not always.

We are only aroused when the baby becomes starved and dehydrated. When this occurs the chances of recovery are lessened when surgery has to be resorted to.

After it has been decided that an organic lesion is present, it is necessary to go further and attempt to decide what type of lesion is present—pyloric stenosis or atresia of the bowel.

This differentiation will help the surgeon to outline his operative procedure.

The differentiation can usually be made by comparing the above facts.

THE IRRITABLE COLON—A COMMON CONDITION, COMMONLY MISUNDERSTOOD.*

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Chronic or recurrent irritability of the large bowel is a cause of symptoms in at least 40 per cent of the individuals consulting a gastroenterologist. It may be due to organic disease, is not infrequent in persons who take purgative medicines regularly or to excess, but often is of purely neurogenic origin. The last type is so frequently misunderstood in that it is treated as on organic dis-

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ease, that I feel justified in discussing it. It has been described by a number of authors under the names unstable colon, spastic colon, mucous colopathy, colitis and mucous colitis.

In order to appreciate the condition, it is necessary to recall something of the physiology of the colon. The right half of this organ has some digestive function, largely through bacterial agencies and a definite absorptive power especially for water; the left half is probably largely a storage tank which normally empties itself at regular intervals, usually, but by no means always, every twenty-four hours. It is important for the profession to remember and to pass on to the laity the fact that the colon is not a cesspool, in spite of the fact that only the left half at most empties when a normal stool is passed, and that the material in the right half is then prepared for a subsequent stool.

This colonic function is the result of a well coordinated nervous and muscular activity leading to peristalsis and anti-peristalsis in orderly fashion. Various factors can easily upset this mechanism, the simplest of which is a purgative. This acts as an irritant which leads to complete evacuation of the bowel. It is readily seen that repeated purgation will cause chronic irritation of the colon with mucus formation, spasm and so-called peristaltic unrest. Purgation is then a common cause of irritable colon.

This irritability may be induced by infectious diseases of the colon, by new growths, by polyps and diverticuli and a number of other conditions. In this paper, however, we are primarily interested in those cases where the condition is of neurogenic origin. It is seen most commonly in those individuals of vagotonic type who live under nervous stress and strain as a result of their work, their domestic and social worries or other things which tend to upset their nervous economy. They cannot relax generally and their colons share in this tension. They become constipated as a result, or, as is not infrequently the case, the constipation alternates with diarrhea. They take purgatives or enemas to empty the colon, and consume roughage, such as bran, to increase the intestinal bulk. These measures initiate a vicious circle which may go on indefinitely and often requires the utmost skill on the part of the physician to break.

A small percentage of these patients may have definite allergic sensitiveness to certain foods which may be the cause of colonic irritability, but skin protein tests are usually quite disappointing and the elimination diets must be tried to determine the offending food.

Vitamin deficiency and a disturbance of calcium metabolism have been suggested as causes of the condition but neither of these has appeared to be important in the etiology. Chronic inflammation of the appendix, the gall-bladder or the pelvic adnexa have been thought to reflexly disturb the nervous mechanism of the colon in certain cases, but it is rare that operation for such organic diseases does more than bring about temporary relief of the symptoms.

Constipation is probably the most common complaint with this disorder. It may alternate with diarrhea and may be associated with a sensation that the bowel does not empty itself after a stool. Not infrequently, especially in women, evacuation of the bowel is followed by a feeling of general weakness and depression. Colicky pain along the course of the colon is a common complaint. It may be mistaken for that arising from organic disease, but is usually more diffuse and the patient's description of it is apt to be vague. The pain is almost invariably increased by purgatives or enemas.

When the individual is constipated, the stool when observed is small and in hard, round segments; it is passed with difficulty and usually with strings of tough mucus. On several occasions I have had patients bring these strings to me insisting that they were portions of tapeworms. Actual mucous casts of sections of the bowel may at times be passed. During other periods the individual may have frequent desire to defecate with the passage of small fecal content and jelly-like masses of mucus. Purulent material is never passed in the neurogenic cases and blood is rarely seen in the stools unless there are local bleeding areas about the rectum or anus.

The general evidence of disturbance of the nervous system is striking in the majority of the cases. Many present the picture of an anxiety neurosis; they have cardiac irregularities; vasomotor disturbances, such as faintness, numbness in the extremities, clammy perspiration; and not uncommonly arthritic manifestations are present. In advanced cases their minds seem to "revolve about their belly buttons." I recently saw in a neurotic negro man the remarkable ability to induce excessive peristalsis

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with loud coloptage purely voluntarily on his part. These individuals develop many false ideas about themselves, their diets and the terrible consequences if the bowel is not emptied daily.

A complete medical and environmental history should be taken in each case as this is extremely important. Physical examination will reveal the neurotic, often visceroptotic type of individual in whom the spastic, sensitive left half of the colon is palpable. Proctoscopic examination of the accessible colon often shows spasm of the lower sigmoid, some engorgement of the mucosa and excessive mucus secretion, but is especially valuable in ruling out organic disease. Examination of the stool microscopically is helpful to eliminate the possibility of parasitic infections. Careful Roentgen ray examination is of particular help and may include a barium enema as well as a barium meal. Of the two, more accurate information can usaully be obtained from the latter. The findings may vary all the way from slight spasticity of a portion of the colon to almost complete obliteration of a section of the bowel giving the "string" shadow. Not infrequently dilatation of the proximal colon with spasticity of the distal may be found.

In the treatment of irritable colon the two important principles are: First, a thorough understanding of the underlying etiology on the part of the patient, and the outlining of a therapeutic regime which will allay the irritation present and prevent, if possible, its recurrence in the future. Sufficient time should be taken in conference with the patient to give him a general understanding of normal bowel physiology, an insight into his own nervous make-up, and the importance of extraneous nervous excitements which enter into his daily life. He should be made to comprehend the necessity of avoiding irritant purgatives and irrigations, and his mental processes should be steered away from his "belly button." Time spent in this psychotherapy will accomplish a great deal more, especially for the future, than any dietary or medical program prescribed.

These patients are very apprehensive about themselves and it is important to avoid using medical terms which to them suggest organic disease. I have seen a number of them who have been told that they had "colitis." To them, this means inflammation of the bowel, and it has been particularly difficult to convince them that their symptoms were purely of nervous origin. Many of them are tall and slender and naturally have low-lying abdominal viscera, but to give them a diagnosis of visceroptosis usually aggravates their "belly consciousness."

The diet to be ordered is of the low residue type. The essential point is the avoidance of coarse foods containing fiber, skins, seeds or gristle. The patient must omit salads, celery, tomatoes, cucumbers and pineapple. Many of the green vegetables contain too much undigestible cellulose, as do raw fruits, such as organges or grapefruit. Such things as raisins, nuts and jams full of seeds should not be allowed. Meats may be eaten freely provided they are tender and well cooked. At times a definite hyperacidity syndrome of grastric symptoms is present which will be benefited by food, such as milk, between meals and at bedtime, and the avoidance of acids, highly seasoned foods and excessive sweets. All drinks containing caffein should be omitted and tobacco smoking should be cut to a minimum. The use of "tonics" containing strychnine and nux vomica is illogical and practically always aggravates the symptoms. Bran, either plain or in cereal or bread, should be absolutely interdicted. If a definite food allergy is discovered, the proper deletions from the diet may be made, but it is important that the patient follow the doctor's instructions as to diet and not one of the numerous quasi-medical lists which are so readily obtainable.

Opinion is somewhat divided as to the value of the anti-spasmodics but in my opinion belladonna in moderate dosage is helpful. Some of the newer, and incidentally more expensive, anti-spasmodics have not proved as effective. Frequently general sedation is desirable. This may be accomplished with proper dosage of bromides or one of the barbiturates. A few days of rest in bed or a short vacation may be quite helpful in the beginning of treatment.

For the constipation, it is surprising how often relaxation and giving the bowel a chance to function normally will correct the condition. Mineral oil alone or in combination with agar-agar (with no added laxative) should be taken in regular dosage. In the early stages it may be necessary to employ minimum and decreasing doses of cascara to establish evacuation, always trying to avoid the passage of a loose stool.

In recent years several articles have appeared in

the literature advocating the use of autogenous vaccines made from the stools in the treatment of these patients. I have not used these but have seen two patients who had taken numerous injections with absolutely no relief of symptoms which could be attributed to the treatment. If my premise is correct, this treatment should at least have a bad psychological effect since in the minds of the laity vaccines are used for the cure of infections.

Colonic irrigations of any kind should be studiously avoided and if an enema seems necessary either normal saline solution or warm oil should be used. I join the proctologists in deploring the too frequent use of soapsuds or other irritant enemas. Local irritant conditions about the anus should be corrected by the proper surgical measures but I warn you that disappointment is in store for those of you who promise relief of the colon symptoms by removal of a chronically diseased appendix or gall-bladder.

From my own experience and that of others, this type of colon disturbance does not seem to predispose to the development of chronic ulcerative colitis, nor are these subjects any more likely to have malignancy of the colon than do the average individuals. It is rather surprising how often elderly people are seen with a long history of colon symptoms.

In conclusion, I wish to emphasize that with proper care in the study of the patient a positive diagnosis of neurogenic irritability of the colon can be made, and if the appropriate therapeutic procedures are employed these chronic sufferers can be made infinitely more comfortable. That an absolute cure cannot be promised in all cases is obvious. Their inherited unstable nervous make-up cannot be replaced nor can we eliminate all of the unfavorable nervous influences, but a good deal can be done toward making the patients live in harmony with themselves and their environment.

Professional Building.

DISCUSSION

DR. OSCAR L. HITE, Richmond: I think Dr. Davis has brought us today a very practical and helpful paper, one extremely helpful, I think, to those physicians practicing in the country, as well as to the men in the cities. There is nothing new that I might add to what he has said. The only thing I could hope to do would be to emphasize some of the points which he has talked about already.

The condition is more frequently found in the female than in the male, being estimated by different men to be about three to two—or some men say as high as three females to one male.

Now, I might say a word about the diagnosis. Dr. Davis made mention of the method of giving a barium enema as well as a barium meal. I think a barium enema is of very little help, because usually it is put in under some pressure and the colon, unless there is a great amount of spasm, is outlined very well on the X-ray film. On the other hand, if a barium meal is taken and allowed to go into the colon in normal fashion, you will find the erea of spasm outlined.

Another thing; if you question the patients about the character of their stools, they will tell you about the ball-like appearance, hard knots, lump-like in character. Dr. Davis spoke of the great amount of mucus these patients pass. About a year ago a patient of mine who was down at the beach called me over long distance and said he had passed a piece of his bowel and said he was going to send it to me. He sent it, special delivery. When it came to me the next day it did smell like a piece of his bowel. It was put in water and it became sponge like in appearance. When it was teased with applicator, it was mucus. This particular patient has a diverticulitis and has a good deal of pain on his left side and also has colitis.

As to treatment, I find the majority of these cases coming to me have been given no instruction whatever as to habits and little as to diet. Now, the majority of these people respond to every impulse they have for evacuation of the bowel. Doctors, as a rule, do not instruct them as to their habits, and they are no more than animals; they respond to every impulse of the bowels to move. If these people are instructed to try to resist some of these impulses and try to have only one movement of the bowels a day, they get along beautifully.

I have found tannin agar to be helpful in this type of case. It was devised by Dr. Max Einhorn, with whom I was associated. It is impregnated with a grain of tannic acid to the dram, I believe. He has used it over a period of years and it has proven beneficial in cases with frequent stools.

DR. J. W. HUNTER, Norfolk: I should like to ask Dr. Davis whether he has had any experience with barium sulphate, using it twice a day?

DR. BEVERLEY R. TUCKER, Richmond: I think there is a very similar condition to the one that Dr. Davis so excellently discussed, and I should like to ask Dr. Davis to let us know what he thinks about it, when he answers this discussion. That is, a good many people, I find, have a somatic delusion about their bowels, and, although their bowels are perfectly normal, while that delusion is on, they can get up a diarrhea or a constipation. I know a woman who came in with a disability of her arm. We congratulated ourselves upon getting the arm well, and then she developed a colitis, so to speak. We treated her for that and got that all

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right. Then she developed a nervous condition, and every time an automobile came within twelve blocks she would nearly jump out of bed. We treated her, and that got all right, and then she could not see. We remedied that, and then she got back on the bowel. (Laughter.)

DR. DAVIS, closing discussion: Dr. Hite brought out one point which I think should be stressed. If my premise is correct in regard to these cases, the general practitioner is the man to handle them, because he is in the position where he can discover the underlying nervous stresses and strains. The patients are not so apt to go into these with a strange doctor whom they only see for a few hours. On the other hand, a thorough diagnostic study will often correct some of their misinterpretations and put them in better position for the family practitioner to guide them to health.

I have used barium sulphate in some of these patients, and occasionally it works very well as a soothing agent. A difficulty, however, is that it tends to cause fecal impactions.

Dr. Tucker knows a great deal more about somatic delusions than I do. His patient is exhibiting symptoms suggesting organic disease probably as an outlet for some severe psychic shock. I am sure Dr. Tucker has discovered the background for this.

DR. TUCKER: Her sweetheart died. (Laughter.)

DR. DAVIS: It seems to me, then, that the thing for Dr. Tucker to do is to find her another sweetheart. (Laughter.)

STRIPS OF FASCIA LATA AS SUTURE MATERIAL IN THE REPAIR OF THE MORE DIFFICULT ABDOMINAL HERNIAS.*

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For practical purposes abdominal hernias may be divided into two groups: (1) those of simple type, in which a very high percentage of cures follows operative treatment, and (2) the group in which satisfactory results are not obtained by the ordinary forms of treatment. For some years I have been especially interested in this latter group, and I am satisfied that, after an extensive trial of living sutures taken from the fascia lata, better results can be obtained than with any other technic.

The greatest objection to the method in the past has been the necessity of making a long incision on the outer aspect of the thigh and the extra time added to the operation. I am now able to obtain all the tissue necessary through an incision not more than 2 inches (5 cm.) in length, with its central point directly over the lower end of the tensor fasciae femoris muscle, by the use of a fascia stripper (Fig. 1). This stripper is made of two steel tubes, one of which is slightly longer than the other, has a handle at one end and an opening close to the other, and fits inside the other tube. This outer tube has a cutting edge at its lower end. After freeing up, through the small incision on the upper outer aspect of the thigh, a piece of fascia sufficiently wide to make as many sutures as I expect will be necessary for repair of the hernia, it is inserted through the opening in the end of the stripper; by careful manipulation this strip can be freed down to the insertion just above the knee. The cutting end of the outer tube is used to cut the strip free. The tissue thus obtained can be divided into as many strips as necessary by simply tearing it in a longitudinal direction, as it splits very readily but is exceedingly strong on a straight pull. Experimental work done by Gallie and LeMesurier more than ten years ago definitely proved that such strips live unchanged when buried in the tissues in such a way as to receive an adequate supply of lymph. They also were able to prove that such strips neither contracted nor stretched and became firmly adherent to all tissue through which they passed. Each strip is threaded on a thick, short, curved needle and tied to it to prevent frequent rethreading. The other end of the fascial strip is tied firmly with silk or chromic catgut and, after making one or two stitches into the tissue close to the hernial opening, the needle is carried through the suture, close to the ligated end, to form a slipknot and firmly anchor the suture (Fig. 2).

*Read before the Seaboard Medical Society, Kinston, North Carolina, December 4, 5 and 6, 1934.

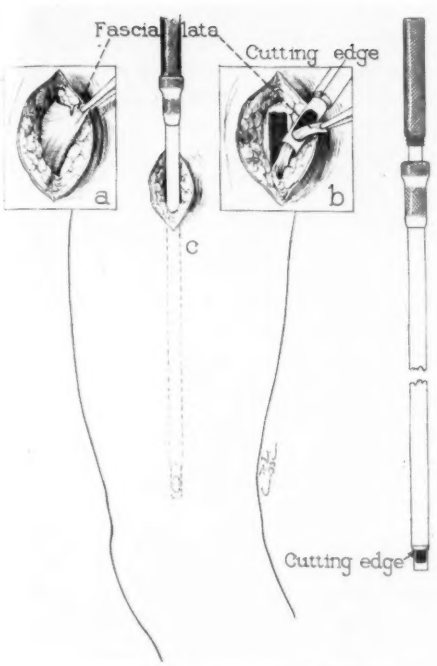

Fig. 1.—Instrument for obtaining fascia lata in use.

In inguinal and other smaller hernial openings, no other suture material is used; in the larger post-operative and umbilical hernias, however, I first like

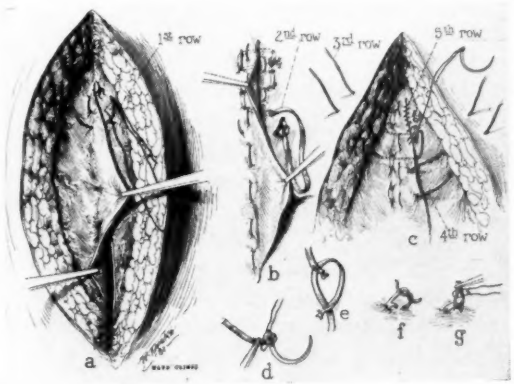

Fig. 2.—Technic of using fascia lata in repair of post-operative and umbilical hernias. a, plastic over-lapping of full thickness of scar tissue and insertion of first row of catgut; b, reinforcing, using living sutures taken from fascia lata; c, further reinforcing with another row of catgut and silkworm gut sutures; d, tying sutures to prevent frequent slipping from eye of needle; e, method of joining one suture to another; f and g, satisfactory method of ending suture.

to close the opening by carrying one side beneath the other and fixing it there with a single strand of chromic catgut simply because it is easier to work with (Fig. 3). The closure is completed by reinforcing with running mattress sutures of living tissue and, if advisable, reinforcing with other living sutures running at right angles to this first row in much the same way that yarn is used in darning a hole in a sock. The other side of the hernial opening is now imbricated on top of the darned area and firmly fixed with other living sutures. By this method, I am sure we are able to effect cures that were not possible with any foreign suture material. Any good anesthetic agent is satisfactory, although I believe spinal anesthesia of special advantage in most cases on account of the complete relaxation, with the resulting minimal amount of trauma to the protruding bowel. In large hernias this is of great importance, as the tension within the abdomen is definitely increased after the closure.

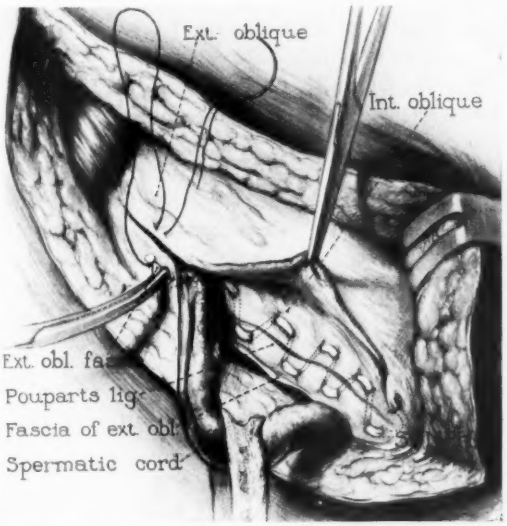

Fig. 3.—Living sutures taken from edge of external oblique muscle. If this is not satisfactory, strips of fascia lata can be used.

In cases in which there has been extensive dissection in the subcutaneous tissue, it is advisable to obliterate the space as much as possible, by either silkworm gut or catgut sutures, and to provide drainage by a large tube for forty-eight or seventy-two hours. The use of a Scultetus bandage is advisable until the patient is allowed out of bed, which, as a rule, is in about three weeks; a well-fitting abdominal binder should then be worn when the patient is up, especially if he is obese.

Most of the large hernias are in cases in which the patient is over-weight, and the question always

comes up as to the advisability of reducing the weight before advising surgery. If the patient lives close by and is having comparatively little trouble, and there are no dangerous symptoms from the hernia, a slow reduction in weight and the wearing of a satisfactory truss of some type is probably advisable, but my experience with attempts at reduction at home have not been satisfactory. Unless 50 to 100 pounds (22.7 to 45.4 kg.) can be taken off, it is of little help, and such reduction should be accomplished in several months or a year. Rapid reduction, or reduction away from home is not satisfactory. With the first method the patient's strength and resistance are markedly reduced, and by the second method the patient is put to considerable extra expense. By using spinal anesthesia and living sutures, I now feel that all patients who are fair surgical risks can safely be operated on with little delay in pre-operative preparation and with a good prospect for cure.

In cases in which it is necessary to remove a wide strip of fascia lata, the possibility of a muscle hernia developing has to be considered. This is not a serious objection to the method, however, as such a hernia does not interfere with normal function and, as a rule, is absolutely symptomless; it is never necessary to repair it. Furthermore, for all but the extremely large hernias, enough fascia lata can be removed without hernia developing. As a protection against dissatisfaction later, all patients should be advised of the possibility of a bulging on the side of the thigh before doing this type of surgery.

SUMMARY

By the use of strips of fascia lata as suture material for the more difficult types of hernia, better results can be obtained than by any other technic. By the use of a fascia stripper, satisfactory tissue can be obtained rapidly and with no inconvenience to the patient. Gallie's and LeMesurier's technic in using fascia has proved satisfactory.

For the larger hernias, a modified plastic overlapping, reinforced with sutures made from fascia lata, has given good results. For obese patients, slow reduction in weight is advisable but seldom necessary.

Spinal anesthesia is of advantage to both surgeon and patient.

There is a little danger of a muscle hernia developing where the fascia has been removed, but this is no contraindication to the operation.

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LEIOMYOMA OF THE GASTRO-INTESTINAL TRACT—A REPORT OF TWO CASES.*

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Although leiomyoma has long been regarded as an important tumor of the uterus, as a gastro-intestinal tumor it has failed to receive the consideration which it merits. By many authorities it has been looked upon as a pathologic curiosity of little importance. In the past twelve or fifteen years, however, the number of reviews of the subject and frequent case reports which have appeared in the medical literature have opened our eyes to the fact that leiomyoma of the gastro-intestinal tract is not such a rarity, and that it may be of grave pathologic and clinical importance.

*Read before the sixty-fifth annual meeting of the Medical Society of Virginia, at Alexandria, October 9-11, 1934.

The following cases have come under the care of Dr. R. L. Payne and myself in the past two years:

Case 1.—Mr. E. S., age fifty-eight, was admitted to the Norfolk Protestant Hospital on July 18, 1932, complaining of a gastro-intestinal upset of one week's duration. On questioning him it was learned that he had experienced a similar attack one year previously, at which time he suffered severely with epigastric pain, nausea, distention and diarrhoea. During this attack he had five or six loose stools a

day and often passed blood and mucus. After five or six days the attacks subsided and he enjoyed good health for a period of nine months. Following this he had a second attack which again cleared up spontaneously after four or five days.

Just prior to his admission to the hospital he consulted a clinician because of a third recurrence. At this time he was having four or five stools a day but was not passing blood or mucus. He had lost about fifteen pounds. The cardiac, respiratory and genito-urinary history was essentially negative.

His past medical history was significant in that he had had two previous operations. An appendectomy had been performed fourteen years previously, and four years later Dr. Payne had performed a cholecystectomy and posterior gastro-enterostomy for cholecystitis and duodenal ulcer. Following this operation he had remained well until the onset of the present illness.

Physical examination revealed a well-developed, well-nourished, white male who appeared to be about fifty years of age. Examination of the head, neck and thorax was essentially negative. On the abdomen were two scars—a McBurney and a right rectus scar. There was no distention or rigidity. The patient complained of slight discomfort on deep palpation over the mid-epigastrium, and an indefinite mass was elicited in the same region. Rectal examination was negative except for bleeding hemorrhoids. The extremities were negative.

The blood Wassermann and urinalysis were negative. The red blood count was 4,510,000 and the hemoglobin was 86 per cent. There was a leucocytosis of 10,200, with 67 per cent polymorphonuclear leucocytes. Gastric analysis showed a total absence of free hydrochloric acid. The roentgenologic examination revealed a filling defect on the lesser curvature of the stomach near the pylorus. Though the roentgenologic findings were not typical of a malignancy, a clinical diagnosis of carcinoma of the stomach was made.

On July 20, 1932, an exploratory operation was performed. The duodenum was found to be bound down and distorted by dense adhesions. After freeing these adhesions, a discrete incapsulated tumor about the size of an English walnut was found on the lesser curvature of the stomach near the pylorus. The mass was movable and extended into the gastro-hepatic omentum. While it encroached upon the stomach wall it did not appear to involve the mucosa. The tumor was excised and the defect in the stomach wall closed with a double row of chromic catgut sutures.

Grossly, the tumor was firm and incapsulated. It was nodular and the cut surface was of a pinkish gray color, and moist. It measured about 38x28x28 mm.

Microscopic section proved that the tumor consisted of irregular bundles of smooth muscle fibres, with a very scanty supporting structure of fibrous tissue and blood vessels. In most areas the tumor was quite cellular and the muscle cells were rather poorly developed. Though the nuclei were rod-shaped, with rounded ends, they were larger than normal and stained quite deeply. Only two mitotic figures were found.

The pathologic diagnosis was benign leiomyoma of the stomach. The patient's post-operative convalescence was uneventful, and on the sixteenth post-operative day he was dismissed from the hospital as cured. To date he is still in good health.

Case 2.—Mrs. M. G., age twenty-eight, was admitted to the Norfolk Protestant Hospital on May 8, 1933, because of intermittent attacks of severe right abdominal pain. She had observed a small abdominal tumor which sometimes appeared in the right lower quadrant, though on other occasions it might be located in the mid-epigastrium. The first attack of pain had occurred about five months prior to her admission. Since the onset the attacks had been frequent, sudden in onset, and of various degrees of intensity. They were characterized by abdominal cramps, moderate distention, nausea and occasional vomiting. There was no history of diarrhoea or constipation and the patient had never observed any blood or mucus in her stools. Diet apparently had no effect. The attacks usually subsided spontaneously but she was never entirely free from abdominal discomfort.

Her cardiac, respiratory, genito-urinary and menstrual history was of no significance. During the five months of her illness she had lost twenty pounds of weight.

Upon examination the patient appeared to be well-nourished and well-developed. Her pulse, temperature and blood pressure were normal. Examination of the head, neck and thorax was essentially negative. The abdomen was soft and not distended. A freely movable mass about the size of a small lemon was palpated in the right lower quadrant. Exami-

nation of the pelvis and extremities was negative. The blood Wassermann and urinalysis were negative, and red and white blood counts were normal. No stool examination or roentgenologic investigations were made.

Because of the movability of the tumor, the absence of any change in bowel habit, and the fact that the patient did not appear to be ill, the intestine was not seriously considered as the source of trouble and a diagnosis was made of intra-abdominal tumor, probably in the omentum.

On May 9, 1933, an exploratory laparotomy was performed. Upon opening the abdomen the tumor was found to be within the lumen of the transverse colon, and immediately behind it was an intussusception involving a freely movable caecum and the terminal portion of the ileum. Obstruction was not complete. By making pressure distal to the tumor, the intussusception was readily reduced and the growth was found to be attached by a pedicle to the caecum just above the ileo-caecal valve. Fortunately the patient had been on a liquid diet for several days and the bowel was practically empty. An incision was made through the longitudinal band of the caecum through which the tumor was exposed. The pedicle was clamped and sutured and the tumor excised. The opening in the caecum was closed by a double row of chromic catgut and the abdomen closed without drains.

For four days following the operation the patient was given nothing by mouth, her nourishment and fluid balance being maintained by intravenous administration of glucose and saline. On the second postoperative day the patient had a severe chill following intravenous glucose, but otherwise her convalescence was uneventful.

Grossly, the tumor appeared about the size of a golf ball, measuring 45x40x35 mm. It was attached to a broad pedicle and was covered with mucous membrane. About half of the mucous membrane was ulcerated. The mass was firm and the cut surface appeared translucent. The microscopic picture was that of a typical leiomyoma. There were irregular bands of smooth muscle fibers supported by connective tissue and blood vessels. The nuclei were rod-shaped, rounded at the ends, and stained deeply. There were no mitotic figures. The tumor was diagnosed as benign leiomyoma of the caecum.

DISCUSSION

Leiomyoma is a neoplasm composed of smooth muscle with a supporting structure of connecting tissue and blood vessels. It is most frequently encountered in the uterus, but may develop in any location where pre-existing smooth muscle is found. While its occurrence in the gastro-intestinal tract has been generally considered rare, the lesion has been often observed in the esophagus, the stomach, the small and large intestines and the rectum, and recent investigations have led to the conclusion it is by no means uncommon.

In 1928 Rieniets made an extensive search, by inspection and palpation, of all stomachs secured at the first 100 consecutive postmortem examinations performed at the Mayo Clinic. From this group he found sixteen stomachs which contained twenty-one tumors that proved to be leiomyomata. Most of them were quite small and ranged from 1/2 to 9/10 cm. in diameter. Because of the unusual frequency of these tumors he ran a second group composed of the next 100 stomachs examined at necropsy and in this group found twenty-two leiomyomata in sixteen stomachs. He then studied all available leiomyomata which had been discovered at routine autopsy examinations from January 1, 1922, to April 1, 1930, exclusive of the cases mentioned above. In this group he found only thirty stomachs presenting thirty-four leiomyomata. The lesions in the group were much larger than the others, ranging from 1/10 to 3 or 4 cm. in various diameters.

Balfour interprets Rieniets' observations as meaning, first, that leiomyomata of the stomach are much more common than the surgeon has considered them in the past, and, second, that in the majority of cases such tumors undergo little if any change in the course of the average patient's life, and therefore go unobserved.

The etiology of leiomyoma is unknown. A number of theories have been advanced, but as yet all remain to be proved. It is derived undoubtedly from smooth muscle, but we do not know the exciting factor which is required to stimulate these isolated groups of cells to proliferation and tumor formation. In the gastro-intestinal tract the stomach is the most common site and males are apparently affected more often than females.

In size, leiomyomata may range from small shot-like nodules, as described by Rieniets, to growths

of quite large proportions. Raiford refers to a myoma of the small intestine the size of a grapefruit or a baby's head. They are usually single but may be multiple, especially in the stomach.

The typical tumor is firm, although the consistency may vary somewhat in proportion to relative amount of muscle and fibrous tissue. The cut surface is pinkish-gray in color and more opaque than normal muscle. It usually appears to be sharply circumscribed and may shell readily out of its bed. It must be remembered that these tumors are particularly subject to regressive changes, and the color and density are naturally altered where hemorrhage, fatty, or hyalin degeneration, necrosis or calcification, have occurred.

The microscopic picture of leiomyoma presents intertwining bundles of smooth muscle supported by varying amounts of stroma which is composed of fibrous tissue and blood vessels. The smooth muscle, however, always predominates. The tumor cells are usually shorter and thicker than the cells of normal muscle, the cytoplasm tends to be less acidophilic, and the nuclei, though rod-shaped with rounded ends, may be larger than normal and richer in chromatin. In the more actively growing tumors there is an increase in the neoplastic character of the cells. They are less well differentiated, may be quite large, and in definitely malignant leiomyomata the muscle cells may closely resemble spindle cells. In this type the connective tissue stroma is very scant, large giant cells are formed, and mitotic figures are usually abundant.

While leiomyomata are usually benign, a certain percentage are malignant. Here, again, arises the old question. Are a certain per cent of these tumors malignant from their beginning, or do benign leiomyomata sometimes develop malignant characteristics? According to Ewing, Delfield and Pruden, and McCallum, either may be the case. Ewing states, "From the uterus, stomach, esophagus and intestines this usually benign tumor has developed extensive local and secondary growths and proved fatal. This event has occurred in the later period of the growth or after extirpation of tumors which had long remained benign, so that it would appear that a malignant change has taken place in the character of the tumor." There are also reports of cases of the type reported by Melneck, where apparently benign leiomyomata have given rise to metastatic lesions, and both original and metastatic lesions failed to present the necessary criteria for a pathologic diagnosis of malignancy. Ewing states, however, that he has never seen a case of this type where careful study failed to reveal some divergence from the characteristic picture of benign leiomyoma.

Occasionally the differential diagnosis is difficult since a rapidly growing malignant leiomyoma may closely resemble a spindle cell sarcoma or cellular fibroma. It is the type which is so frequently diagnosed leiomyo-sarcoma—a very confusing terminology. It is conceivable that the fibrous tissue cells of the stroma may be stimulated to the degree of producing an abnormal new growth, but in this event the fibroblasts would predominate rather than the muscle cells, and the tumor would be classed as a sarcoma. The following criteria are often helpful in differentiating the one from the other: Muscle cells are usually sharply outlined and have rod-shaped nuclei with rounded ends which lie within the cell bodies. The nuclei of fibroblasts are definitely spindle shaped, shorter, and lie on the surface of the cell. Muscle does not show the definite wavy striation characteristic of connective tissue, and is apt to contain more nuclei in a given field. Muscle cells usually terminate in a definitely pointed extremity, while the tip of the fibroblast is apt to break up into an arborization of fine fibriles. Finally, the staining properties of the cell may be of considerable aid. Muscle is more strongly acidophilic than connective tissue, and therefore takes a more intense red color in sections stained with eosin; Van Guson's stain shows muscle yellow, connective tissue red. Mallory's stain shows muscle red and connective tissue blue.

Gastro-intestinal leiomyoma may be intra-luminary or extra-luminary, depending chiefly upon the layer of muscle from which they are derived. For example, a tumor arising from the muscularis mucosa would more than likely be intra-luminary and pedunculated, whereas one springing from the external layer would develop between the layers of the mesentery, or, if opposite the mesenteric attachment, would by stretching the peritoneum become subserous.

The fact that many leiomyomata of the gastro-intestinal tract remain dormant and never produce symptoms worthy of note has already been emphasized. On the other hand, they are capable of a wide variety of complications, depending upon the

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location and size of the growth as well as the degree of malignancy. Submucous growths, for instance, are particularly prone to ulceration and hemorrhage. The latter may range from a slow gradual oozing to a sudden massive hemorrhage and death. According to Wolfer, Rieniets, Balfour, Dixon and Steward, Raiford, Melnick, Rankin and Down, such tumors may readily be the cause of gastro-intestinal bleeding of obscure origin, which may lead to severe secondary anemia and eventually death. Furthermore, ulceration may result in perforation and peritonitis, as it did in a case reported by Cattell and Woodbridge.

In the stomach, a submucous pedunculated growth may produce an intermittent pyloric obstruction, and in the small and large intestine it is not uncommonly the cause of intussusception.

Subserous growths are rarely pedunculated, and Wolfer states that they may vary in size from that of a cherry to that of a mass filling the entire abdomen. Adhesions often develop, as the result of which the tumor may become fixed to the viscera, or to the abdominal wall, thereby increasing the possibility of intestinal obstruction. In the case of the small intestine, angulation may be produced by the weight of the tumor, even in the absence of adhesions. Where the growth is of the mixed type and encroaches upon the mucosa, ulceration and hemorrhage may result, as it does in the intra-luminary type.

Thus we see that even though leiomyomata may be benign, they may be the source of grave complications and should therefore be considered in the diagnosis of any case presenting vague abdominal symptoms. This is particularly true where a history of intermittent attacks of intestinal obstruction is obtained, or where the case is complicated by gastro-intestinal hemorrhage of obscure origin.

More often than not the tumor is not palpable, in which case the roentgen ray is of great value in arriving at a diagnosis. The treatment is surgical, the technical procedure depending upon the immediate complications to be dealt with, the location of the growth, whether or not it is malignant, and in benign cases the risk of the operation *versus* the risk of carrying the tumor.

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Medical Arts Building.

DISCUSSION

DR. CARRINGTON WILLIAMS, Richmond: Dr. Jones has presented a very interesting question to us today, and from the figures which he quotes from the Mayo clinic, one which must be very much more prevalent than is usually seen.

However, I can't believe that the figures are correct which state that sixteen to twenty per cent of all stomachs would show these particular tumors. They must be very much less frequent than that. I think that if sixteen per cent of all people had this tumor, even though very small, it would be much more frequently seen in general surgical practice. My impression is that it is very rarely seen. In fact, all benign tumors of the stomach and all tumors of the small bowel are very rare conditions.

Dr. Jones' two cases illustrate two of the conditions which result from the presence of these benign tumors his first being a deformity of the pyloric end of the stomach, and the second case an intussusception resulting from the tumor in the bowel.

He has called attention to the other facts which also may suggest the presence of this tumor, such as hemorrhage and pyloric obstruction, intestinal obstruction, due to the bulk of the tumor, and that intestinal obstruction when not complete gives rather vague indefinite abdominal pain, and of course when complete the typical picture of intestinal obstruction.

It also may be diagnosed, or rather the general condition may be diagnosed, by a palpable tumor, because, when these tumors are sub-serous and do not encroach on the lumen of the bowel, they may grow to a very considerable size.

Now, in Dr. Jones' second case, he tells us that the patient lost twenty pounds in weight. I think that is very unusual in this condition, except in those cases of hemorrhage and pyloric or other intestinal obstruction.

I was also interested in Dr. Jones' discussion of the

question of the malignant muscle tumor, and whether the malignancy was the result of a change from the primary benign tumor or whether it started out as a primary malignant tumor. My impression is that the pathologists in recent years have rather inclined to the idea that a malignant tumor has always been a malignant tumor, and the change from benign to malignant is very rare. I also have the impression that there is a change the other way now, and that the pathologists are swinging toward the theory that has been held for so many years that benign tumors may become malignant.

The few cases of malignant leiomyoma have certainly given a history that is consistent with that of a benign tumor over a period of many years, and my personal impression is that benign tumors can become malignant, and need not necessarily start as malignant tumors.

I think Dr. Jones has called our attention to a very important subject, and if the frequency comes anywhere near the figures which were shown in the Mayo Clinic report, it is something that we should be much more on the look-out for than we usually are.

DR. CHARLES R. ROBINS, Richmond: I want to report a case of leiomyoma of the small intestine.

As I understand it, these cases are very rare. The interesting point about this particular case is that the patient was an elderly gentleman, with a history of what appeared to be an incomplete obstruction. He was suffering a good deal of pain, and most of his trouble was in the left lower abdomen. It looked like a case of malignancy.

He was operated on, a marked intussusception was found, and a large part of the small intestine was distended. That was relieved, and the patient was supposed to be cured.

However, he did not get well, and continued to complain, and he began to get worse again. At the second operation, we found one of these benign tumors, which had evidently been the cause, in the small intestine. This was about the size of a horse chestnut, and the mucous membrane of the small intestine was eroded at the point of contact, but there was no erosion on the tumor itself.

This tumor was certainly benign. It was interesting because it occurred in an old man. How long it had been there, we don't know. But, at any rate, he got well, and is all right now.

DR. JONES, closing the discussion: I wish to thank Dr. Williams and Dr. Robins for their discussions. I agree with Dr. Williams that the frequent occurrence of leiomyomata in Rieniet's review is startling. However, his report has opened our eyes to the fact that these tumors do occur much more often than we have realized in the past, and I feel sure that a more careful search for them in routine examinations of the stomach and intestine will bear out this conclusion.

A PROSTHETIC OBTURATOR AND PLUMPER.*

JOHN BELL WILLIAMS, PH. G., D. D. S.,
Richmond, Va.

In dentistry the term "prosthesis" means the replacement of missing teeth by artificial substitutes. An "obturator" is an appliance which closes an abnormal opening between the mouth and other cavities. A "plumper" is a device carried in the mouth to fill out the cheeks. To sticklers for scientific terms these three unusual words are necessary to describe a certain piece of dental mechanics. Since unusual words are sometimes misused, this paper will be called the report of a dental plate designed to cover unnatural openings and to restore a sagging cheek.

This replacement was made for a patient referred to me by Dr. S. W. Budd, who kindly wrote the following brief history:

> "In the early summer of 1933, Mr. R., a young man of about twenty years, consulted me concerning a growth the size of an orange involving the left antrum and the hard and soft palate, and also extending out into the cheek. The growth and the infection accompanying it made a sizable swelling which partially closed the left eye. There was considerable bone destruction. The growth had broken through the hard palate and bulged into the oral cavity.
>
> "The patient had been treated surgically by Dr. E. C. Bryce who recognized the condition was malignant and sent him to me for consultation and radium treatment. I obtained some tissue for biopsy and found the growth to be a very active perithelial sarcoma (Fig. 1). I informed the family that I feared little could be done for him but I would give him the benefit of radium treatment.

*Read at a meeting of the Staff of the McGuire Clinic, St. Luke's Hospital, Richmond, Va., October 16, 1934.

"The young man was taken to an evangelist by friends who prayed with him and finally converted him, but for some reason or other the tumor did not disappear as a result of the prayers. Some two or three weeks later he came back to get his radium treatment. For the first treatment I gave him 2,400 milligram hours with capsules (screened by brass and rubber) placed into the antrum, and with needles widely distributed over the growth. A month later I gave him 1,800 milligram hours with capsules placed into the antrum and again screened with brass and rubber. He was then referred to Dr. E. L. Flanagan who gave him an extensive series of X-ray treatments over a period of three months. About three months later I

Fig. 1.

saw the boy after the infection and reaction had subsided and the growth apparently had disappeared. A large portion of the hard palate on the left side had sloughed out and he talked like one with a cleft palate. I referred him to Dr. J. B. Williams for a correction of the deformity of the mouth.

"The patient informed me some six or seven months after his recovery that his prayers and not the radium had cured him. I felt rather keenly that we should be denied the credit of this brilliant result."

The dental examination revealed the presence of all of the lower teeth and all of the upper teeth except the left bicuspids and molars. The opening through the palate was a very large one, while the opening from the vestibule of the mouth into the

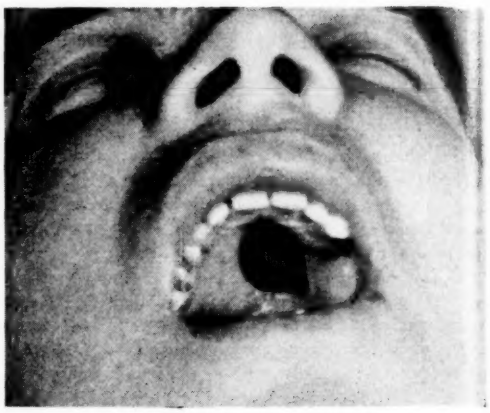

Fig. 2.

antrum showed the destruction of a portion of the floor and the entire outer wall of that cavity. Fortunately, a bridge of strong healthy tissue separated these openings (Figs. 2 and 3). Until a short while ago it was difficult to obtain a dental impression of such deformities, but with the use of dentocoll, a new material with an elastic base, the technique is now easy and simple. The dentocoll when warm is soft and can be forced into openings. When it

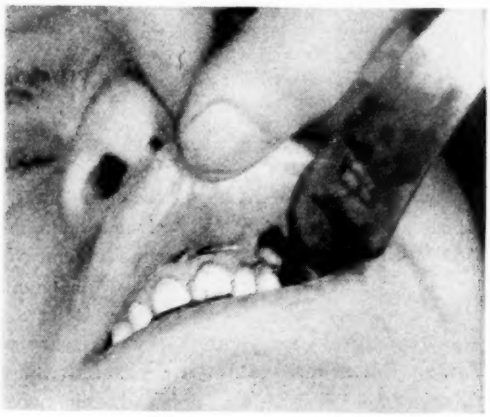

Fig. 3.

cools it becomes hard and makes an accurate mold of the space it occupies. Owing to its elasticity it can be compressed and removed without breaking. It flies back in place like a rubber band and it shows all under-cuts and other inequalities of the surfaces. From this negative a positive cast or model of the mouth can be made in plaster.

An appliance constructed on this type of cast was used in the case under discussion (Fig. 4). It consisted of a hard rubber plate, replacing missing teeth, covering the entire hard and a portion of the soft palate, and artificially reproducing the walls of the maxillary sinus. Stabilization and retention were secured by the use of rubber spurs and gold bands around the natural teeth (Fig. 5).

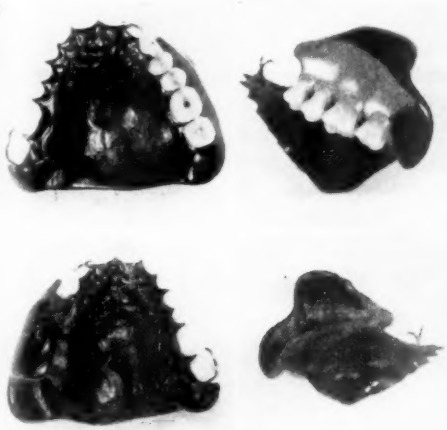

Fig. 4.

Fig. 5.

Within two days after the patient began wearing the plate the outline of his face became much more normal and he was able to eat without discomfort and to speak with an approximately normal voice.

REPORT OF A CASE OF SIAMESE TWINS AT FULL TERM.

E. L. JOHNSON, M. D.,
Bedford, Va.

On the night of October 13, 1934, at 2 A. M., I was called to attend Mrs. P., white, who lived in an isolated and mountainous section of the county, a distance of about twenty-five (25) miles from my home.

On arriving at the home about one and one-half hours later I found Mrs. P. in the first stage of labor. She was a woman of about 125 pounds—normal weight—and twenty-seven years old. Her history revealed the fact that she had given birth three years ago to a normal child with no unusual trouble during pregnancy or at the birth of the child; her history also recorded a miscarriage about eighteen months ago after two months pregnancy.

As soon as possible I made the usual preliminary examination. I found a very large abdomen and what was apparently an unusually large fetus. I was unable to detect but one fetal heart sound. On rectal examination, I found the os to be slightly dilated and the head presenting normally. I administered two sodium amytal capsules and the patient progressed normally during the first stage of labor.

At 6 A. M., I made a second examination and found the os completely dilated and the second stage of labor begun. At this point I started giving chloroform, and in a short time a head was delivered. In attempting to rotate the body in order to expel the shoulders, I encountered great difficulty in rotating it in either direction. It was at this point I realized that I was facing an abnormal situation.

While still attempting to deliver the posterior shoulder I found there were two arms instead of

one, and, in following the arms, they both seemed to be attached to the same body. On examination of the anterior shoulder I found only one arm. Thinking perhaps that I was dealing with an ordinary case of twins, I pushed the posterior arms back as far as I could, then worked the anterior arm and shoulder from under the pubic bone. I then made traction with my hands on the delivered head, but was unable to move it in any direction. I then proceeded to work the posterior arm and shoulder out, but with all the traction I was able to exert on the head I was still unable to deliver it. At this point I rotated the body back to the hollow of the sacrum; then, by pushing my hand up by the side of the child's body, I was able to grasp a leg. By bringing this down, the head and arms of the child having already been delivered, the rest of the body was easily expelled. The delivery of the second child was practically easy. By grasping the feet and doing a version the body and head were delivered without difficulty. The head of the first child was the first to be delivered, while the head of the second child was expelled last. To my surprise and utter amazement, I found that birth had been given to two well-developed girl babies, except for the fact that their bodies were joined together in front, from the clavicles to the umbilicus. Their combined weight was 14 pounds.

Looking at them from the front, there seemed to be only one body, but from the rear they had separate backs and normal spines. Both heads were well developed.

Their buttocks, legs, arms and genitals were normal; in fact, I would call them two perfect specimens of newborn babies, except for the fact that they were united. These children were alive at the beginning of labor, but, owing to the great difficulty experienced in delivering them, they were still-born.

You may imagine my predicament at this lonely mountain home, twenty-five miles from the nearest hospital or doctor and no one to help except an inexperienced sister of the patient who grew very hysterical as the case progressed. I was fortunate in the fact that the patient was not lacerated, but I had to contend with an adherent placenta which gave me almost as much trouble as the delivery of the children. I am glad, however, to be able to say that, in spite of the prolonged and exhaustive delivery, the patient made an uncomplicated recovery.

REVIEW OF PAPERS READ AT MEETING OF THE AMERICAN SOCIETY OF CLINICAL PATHOLOGISTS.*

REGENA COOK BECK, M. A., M. D.,
Richmond, Va.

The thirteenth annual convention of the A. S. C. P. was held at Cleveland, Ohio, on June 8th to 11th.

It will be impossible to do justice to each paper presented at the meeting or to review all of them, so I have only reviewed a few of the things which seemed outstanding to me.

Drs. Kracke and Parker, of Atlanta, presented a paper on the etiology of neutropenia with particular reference to drugs containing the benzene ring. Their experimental work and research indicates that

*Read before the Stuart Circle Hospital Clinical Club, November 14, 1934.

acute fulminant neutropenia and probably the chronic types as well are caused for the most part by the administration of easily oxidizable benzene ring drugs—notably amidopyrine, occasionally phenacetine and dinitrophenol. Barbiturates as a class have little or no effect in the production of the leukopenic state.

Dr. Martin, of Norfolk, read a paper on the treatment of malignant neutropenia by injection of liver extract. He reported three cases thus treated. All three patients recovered and were well at the time of reporting. One of them had to continue on a regular ration of liver.

Dr. Hellwig, of Wichita, presented a paper on morphogeographic studies of the thyroid gland obtained at autopsy. He demonstrated that the histology of thyroids of patients from different countries differed greatly and that this difference was manifest from infancy to old age.

Dr. Ikeda, of St. Paul, demonstrated that pneumonia in babies is frequently due to oil aspiration from the food and from nose drops. He presented many cases and demonstrated the oil in the lung tissue by special staining methods.

Dr. Banks, of Indianapolis, presented the work he has been doing in treating lues of the central nervous system. For centuries physicians regarded fever as an alarming symptom. In 1917 it was demonstrated that artificially induced fever was capable of overcoming the ordinarily disastrous effects of syphilis of the central nervous system. In the short period which has lapsed since this discovery, evidence has accumulated which makes it quite apparent that fever exerts an adverse influence upon the growth of bacteria, diminishes the potency of toxins, favors phagocytosis, and stimulates the development of immune bodies. The inherent dangers of engrafting one serious disease (malaria) upon another as a therapeutic measure naturally led to a diligent search for a less hazardous method of producing the same effect. Comparable results were obtained by other workers following inoculations with the organisms of rat-bite fever, relapsing fever, typhoid vaccine and other foreign proteins. It became more and more apparent that simple fever production was the one factor common to all of these methods. Dr. Banks used a vaccine made from pyrogenic bacilli killed at a high temperature. Wassermann fast patients became two-plus, one-plus, or negative. The majority of his patients returned to work. The results seemed to be comparable with the results of malarial treatment and not so dangerous to the patient.

Dr. Kernohan, of Rochester, demonstrated by lecture and lantern slides the preparation of neurologic material for histologic study. He gave his classification for tumors of the brain and stated that the methods given were incomplete, but, by their use, most lesions of the nervous system could be detected and a correct diagnosis reached. The purpose of the paper was to simplify the methods and to make possible the study of neurologic tissue in any laboratory without the necessity of elaborate equipment or of very complicated staining methods.

Dr. Foord, of Pasadena, presented a case of subacute bacterial endocarditis due to diphtheroid bacilli. Blood cultures on this patient were always positive and although the colonies on the blood agar plates looked like streptococcus viridans, the smears showed the organism to be a diphtheroid bacillus. These diphtheroid organisms were agglutinated in a 1-5000 dilution in the patient's serum. At autopsy a culture was made from the heart's blood. This also showed a diphtheroid bacillus. Sections of the heart valve showed diphtheroid bacilli in the tissues. Dr. Foord continued to subculture and re-subculture the organism, and after many subcultures the diphtheroids changed to streptococci. This demonstrates that there are still many things to learn about the habits of bacilli.

Dr. Osterberg, of Rochester, presented a paper on the arsenic content of the brain in relation to so-called hemorrhagic encephalitis. The hemorrhages are limited almost exclusively to the white matter, the gray matter being practically unaffected. This finding is not usual in any recognized form of encephalitis. The white matter of the entire cerebrum, midbrain, pons, cerebellum, and even of the pyramidal tracts of the medulla oblongata may be infiltrated with innumerable petechial hemorrhages. His microscopic slides showed red cells surrounding the capillaries which were thrombosed and sometimes hyalinized. The red cells were in an excellent state of preservation, without any sign of degeneration or of formation of blood pigment. There was no inflammation present. In places it looked like gross hemorrhage but microscopically it showed small petechial hemorrhages packed together. He says

that arsenic in food as well as arsenical treatment will produce this condition. The patients go into coma from two to five or two to twenty days after ingesting the arsenic or after treatment. Dr. Osterberg suggests that, in the presence of an unexplained gross hemorrhage or of multiple petechial hemorrhages in the white matter of the central nervous system, a chemical investigation for arsenic be carried out, since the presence of relatively large amounts of this element may explain the cause of hemorrhages.

Dr. Kehoe, of Cincinnati, presented a paper on the determination of lead in urine and tissues. He states that the rough tests used in clinical laboratories are worse than useless and especially since a legal angle in these cases is always possible. Dr. Kehoe feels that this test is a chemists problem, with which statement I agree after hearing the paper.

Dr. Tripoli, of New Orleans, gave his diagnostic methods in amebiasis, and the relative value of stool culture as compared with other methods. A pathologist who has been stationed at Panama for some years, and whose name I have been unable to recall, gave as his opinion that the fresh wet and fresh stained preparations were best for diagnostic purposes. He pointed out that with cultures it was still necessary to identify and classify the amoeba. Scrapings from the rectum examined as fresh wet preparations, is the method he uses.

Dr. Kline, of Cleveland, told of further studies made on antigen emulsion preparation for the ball test. This is a test for syphilis. The antigen and patients serum are mixed together in a test tube. Salt solution is added and after shaking the tube a white ball forms, suspended in a clear fluid, in a positive (four-plus) test. This would be a very easy test to read. Further studies are being made by Dr. Kline to perfect a stable antigen.

Dr. Eagle, of Baltimore, described his results obtained with a uniform stable and lyophile complement. Complement is one of the reagents used in the Wassermann test. The serum of guinea pigs is the source of this complement and it is necessary to collect it fresh each time the Wassermann test is run. Ways and means of preserving this reagent have been under investigation for some time. Dr. Eagle has devised a way of rapidly drying the sterile guinea pig serum in an apparatus by means of a vacuum and carbon-dioxide. Sterile water is used to dissolve the dried serum and it is then ready for use. Complement, thus prepared, has remained at a constant titer for five months. This is as long as the tests have run but he feels that it will keep much longer. The very practical application of this experimental work is that immune sera can be prepared in this same way. Immune sera, as they are now prepared, deteriorate very quickly. With this new method the patients will be certain to receive potent sera.

Dr. Heise, of Uniontown, discussed the medico-legal application of isoagglutinins. He presented a case in which he was called upon to determine whether a certain man was the father of a child. In this connection it might be interesting to call to your attention the three new blood groups discovered by Landsteiner and Levine. These new blood groups are designated M, N, and MN. Owing to the fact that the new blood groups form no antibodies in the human blood the mixture of M blood with N blood during a transfusion does not constitute a menace. The newly discovered blood groups are inherited in accordance with definite laws. The determination of the M and N groups alone is sufficient to divert suspicion from a suspected father in 18 per cent of the cases. In combination with A and B groups (i. e., types 2 and 3) the number of positive results are increased to 31 per cent. The fact was emphasized that you can prove non-paternity in 31 per cent of the cases but not in any case can you prove paternity.

Dr. Hodges, of Huntington, W. Va., read a paper on paralytic accidents due to anti-rabic inoculations. He had observed three such cases. He says there is one complication in 3,500 treatments and that children and old people seem immune while intellectual people seem to be the ones to show this complication.

Dr. MacCarty, of the Mayo Clinic, discussed the early diagnosis of cancer of the stomach. He says that any ulcer larger than a quarter is cancer, while the small ones may or may not be. He also states that 35 per cent of all cancers are in the stomach. He says that any patient with the slightest gastric symptoms should have a gastric X-ray; in fact, a routine gastric X-ray every six months would greatly reduce the mortality from gastric cancer. He reiterated that the early diagnosis of cancer rests on the roentgenologist and pathologist and that the patholo-

gist must be able to recognize and distinguish the cancer cells from all other body cells.

Dr. Sweany, of Chicago, read a paper on pathogenesis of lung cancer, stating that this condition is on the increase. He gave as causes: dust, tar, diseases, etc. He said that influenza was the only disease to deserve consideration as, when it heals, it simulates a carcinomatous growth. The cause of primary carcinoma of the lung is probably the same as for carcinoma elsewhere in the body. Radium in radium mine workers is the only definite etiologic agent. The growth originates from the basal cell layer of the mucosa of the bronchus which is the regenerating membrane. This basal cell membrane can grow cells of many different shapes, basal cells, squamous cells, or cylindrical cells. The basal cell gives the most rapid growth and is the most radiosensitive. The squamous cell type is the most amenable to surgery.

Dr. Brines, of Detroit, reported fourteen cases of primary carcinoma of the lung. Seven were adenocarcinoma, four squamous cell carcinoma and three basal cell carcinoma. He terms the basal cell carcinoma, oat cell carcinoma. This latter type formerly was often diagnosed sarcoma. He also stated that all primary carcinomas of the lung are bronchogenic in origin.

Dr. Anna May Young, of Cleveland, demonstrated the rapid pigment appearance in Ohio red-bellied Dace as a test for intermedin. Intermedin is the internal secretion from the middle lobe of the pituitary gland which controls pigment metabolism. She obtained her intermedin by mincing ox pituitary and extracting with NaOH. The test animal, the red-bellied Dace, is a minnow found in certain Ohio streams. Normally the under surface of this fish turns a bright red during the spawning season. Using a tuberculin syringe, she injected under the shin of the backs of these fish .05 c.c. of the extract of intermedin. In less than three minutes their under surfaces were a bright red. Urine from a patient with a tumor of the middle lobe of the pituitary will give a positive test. The urine is concentrated ten times and one-tenth of a c.c. is injected. The urine is very toxic to the fish and, because of the large dose necessary, many die. Experimental work is being carried on to overcome these obstacles. An increase of this hormone is also found in the blood of patients having melanosarcoma and neurogenic sarcoma.

Dr. Bleyer presented a case of intestinal fibromatosis. The condition followed a ruptured appendix. The ascending colon was greatly thickened due to a fibrosis of the outer coat and the lumen was reduced to 5 millimeters. The condition simulated malignancy. Complete removal was necessary because of the obstruction.

While in Cleveland, I visited Dr. Kline's laboratory at Mt. Sinai Hospital to have him demonstrate the Kline test for syphilis. This test, like the Kahn test, is a precipitation test. However, instead of using large quantities of reagents in a test tube, small quantities of antigen and patient's serum are mixed on a glass slide, rotated for two minutes and then the results are read by the aid of a microscope. A four-plus gives an agglutination that looks very much like the agglutination of incompatible bloods. Even the one-plus is easy to read. It is my intention to run a series of these tests and, if the results are as I expect them to be, I will suggest substituting this test for the Kahn test, as I consider the Kahn test in the weaker readings very difficult to read and therefore unsatisfactory. There is always a chance of missing a one-plus Kahn. With the Kolmer Wassermann, a one-plus or plus-minus would not be missed even by a person who had had very little experience in reading them. For this reason most serologists have retained the Wassermann test and used the precipitation tests as a check.

Dr. Exton, of New York, gave a new technic for the sugar tolerance test which cuts the time of performing the test from a two-hour period to one hour. The technic is based on Allen's paradoxical law of dextrose which is as follows: the paradoxical law of dextrose distinguishes sharply between diabetic and every type of non-diabetic animal. Whereas in normal individuals the more sugar given the more is utilized, the reverse is true in diabetes. The procedure of the test is as follows: a fasting specimen of urine and blood is collected. However, previous food taken by the patient has no effect on the test except that the curve is higher, but the character of the curve is not affected. After taking the fasting specimens, the patient is given 50 grams of glucose in 325 c.c. of water, which makes it a 15 per cent solution. This solution, flavored with lemon

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juice and chilled, is easy to drink within one minute. In exactly one-half hour after the glucose solution has been taken, another specimen of blood is taken for a sugar determination. Another 50 grams of glucose in 15 per cent solution is given. In exactly one-half hour another specimen of blood is taken and a specimen of urine. A normal patient will not show any sugar in the urine following this amount of glucose given in this way. In a normal person, ingestion of 50 grams of glucose stimulates the insulin-glycogen mechanism to such activity that the normal organism is then able to deal with any amount of glucose without becoming hyperglycemic. When the second 50 grams is ingested it is better handled than the first. In the diabetic the insulin-glycogen mechanism fails; the first 50 grams of glucose uses up what insulin there is, it is not produced in excess, nor even in a normal amount, so that, when the second 50 grams is ingested, the patient reacts with a distinct hyperglycemia. The curves given by this new test are typical and easy to read.

Beginning with the September number of the *American Journal of Clinical Pathology*, all of the papers read at this meeting will be published in detail. Anyone wishing additional information on these subjects can refer to these journals.

1103 West Franklin Street.

SOME OBSERVATIONS ON THE CARE AND TREATMENT OF DISEASED TONSILS.*

BOOKER LEE, M. D.,
Pulaski, Va.

Diseased tonsils is one of the most common conditions which undermine the health of the human body that we face today and which has to be met and studied by both the general practitioner as well as the specialist.

As diseased tonsils covers such a large field, we will only briefly discuss the symptoms and treatment.

As to their clinical appearances and for matter of descriptive purposes, we will divide the faucial tonsils into several varieties.

When it is of ovoid or almond shape it is usually a sessile, unoffending, pitted body which does not markedly project beyond the plane of the internal edges of the faucial pillars.

They may be partly covered by hypertrophied anterior pillars which it is necessary to draw aside in order to disclose them. These are spoken of as "submerged tonsils." Their retracted state and the valve-like action of the mucous fold over them, preventing the occasional emptying of the lacunar pits, are responsible, doubtless, for the frequency with which patients bearing such tonsils give evidence, by the swollen state of their cervical lymphatics, of their acting as the reservoir for bacterial proliferation, and as the portals of systemic infection.

When projecting beyond the pillars toward the middle line of the pharynx, the wide, gaping mouths of the lacunae are likely to be filled with buccal secretions or food, if they are not occupied by the products of tonsillar inflammation. They may be so large as to meet in the middle line, and when this condition obtains in an especially capacious adult pharynx they are very formidable looking structures.

TREATMENT

A thorough course of medical treatment under close observation should be persisted in at first by using antiseptic and healing throat sprays and gargles, as Tincture Merthiolate, S. T. No. 37, Peroxide, Silvol 10 to 25 per cent, Guaiacol, Tannic Acid and Glycerine, Tincture Iodine 25 to 50 per cent with Glycerine, 25 per cent Ichthyol, or topical applications of 25 to 50 per cent Silver Nitrate two or three times a week.

Before taking up the operative treatment for removal of tonsils, I feel that great judgment should be exercised when advising patients to have their tonsils removed, because it is quite an operation to

*Read before the Southwestern Virginia Medical Society, Radford, Va., September 21, 1934.

subject your patient to, and it would be a great disappointment both to the patient and to the specialist if the desired benefit or results were not obtained.

First, we will take up the local method which is usually contraindicated in patients of a very neurotic type, in cases with valvular heart disease, and in children under fifteen years of age. Where the local method is used, even when the patient is vigorous, strong and healthy and has a very steady nerve, I think psychology plays a very major part, and I always leave it up to the patient whether they want to have their tonsils removed locally or under a general anesthetic.

For local tonsillectomy I usually prefer the following technique: Use a 2 per cent solution of novocaine with fifteen minims of adrenalin to the ounce. Inject just under the mucosa, along the entire length of the anterior pillar, around the upper pole and along the upper one-third of the posterior pillar. Then with my needle I go through the tonsil and inject its posterior surface at the upper, middle and lower pole, which usually anesthetizes the large nerves entering its posterior surface. By this method the tonsils can be removed absolutely without any pain to the patient.

Then with my Leland knives, with blades at right angle to the handle, I free and preserve the anterior pillar around the upper pole and upper one-third of the posterior pillar. With curved tonsillar scissors and the Hurd separator I thoroughly free the upper pole and anterior surface of the tonsil; then I apply the wire snare and allow it to do the remainder of the dissection. By holding a sponge with a little pressure in the fossa for about one minute the small oozing is controlled.

For removal of tonsils under a general anesthetic I prefer to use the Douglas Demoresque tonsillotome which has a fenestrum the size of the tonsil and two blades—a sharp blade, and a dull or hemostatic blade. The most important point with this operation is the technique of thoroughly getting all of the tonsil through the fenestra and leaving nothing but a portion of the mucous membrane and the anterior and posterior pillars; then I shove down the hemostatic blade and lock it good and tight and allow it to remain in place for three minutes, which is the usual coagulation time. By the vise-like pressure of the hemostatic blade the lumens of the blood vessels are sealed together. With my tonsil forceps I grasp the tonsil to keep it from being aspirated or swallowed. Then I come down with the sharp blade which completely cuts the tonsil off.

The advantages of this method are: You can do a completely bloodless tonsil operation; the anterior and posterior pillars are entirely preserved; there is very little resulting edema and traumatism to the surrounding tissues; patients convalesce more rapidly, and it always leaves a good looking throat.

In this operation as well as in the local method great skill is necessary in order that the dissection will completely remove the tonsils and leave no remaining tags or piece of tonsil. Effort should also be made to minimize the post-operative scar tissue and deformity to the pharynx in order to leave the throat in the best possible condition.

CHEMICAL INJURY OF CORNEA IN THE NEW BORN, WITH REPORT OF EXPERIMENTS.

SIDNEY TRATTNER, M. D.,
Richmond, Va.

The importance of corneal scars and the role they play in reducing vision needs no emphasis. It would therefore seem a likely subject for investigation from the therapeutic standpoint. Much work has been done but a search of the literature reveals very little of much value. Most of the work is over ten years old. In fact, the scarcity of the reports in the later literature available was surprising.

The fine recuperative powers of children's and especially infants' corneal tissue are well known. Damage that would be permanent in an adult, with loss of useful vision, is often recovered from in children. None of the literature seen discussed this difference in recuperative power between the infant and the adult cornea. To say that in one instance the tissue is young and in the other old seems

merely to be begging the question. From work that has been done in tissue culture *in vitro,* the indications are that, in some instances at least, tissue cells are potentially immortal; that aging is a biochemical process subject to control.

CASE REPORT OF CHEMICAL INJURY OF THE CORNEA IN THE NEW BORN

W. J., new born, colored, was brought to me by the doctor who had conducted the delivery the previous day, June 25, 1933. The history given by the doctor was as follows: Immediately after delivery he had followed his usual procedure of instilling drops in the child's eyes. In a short time marked edema of the lids developed with evidence of discomfort on the part of the child and finally a purulent discharge. I saw the child when it was about twenty-four hours old. At this time the lids of both eyes were very edematous, the conjunctivae were swollen and deep red, and there was a muco-purulent discharge. Both corneae were badly damaged, no clear corneal tissue being visible. The damaged areas were white, rough and totally opaque, having the appearance of rough moist plaster. The left eye appeared the worse.

A smear of the discharge showed an absence of organisms. Questioning the doctor brought out the fact that he had asked the druggist for a silver nitrate solution of the strength he had been accustomed to use. It was his opinion that the druggist had given him a stronger solution. No prescription had been written so that it was impossible to verify this. The drops were in the possession of the patient's father who was keeping them as evidence since he intended to bring suit. He could not afford the chemists' fee for examination. The result was that it was impossible to determine the exact nature of what had caused the damage. However, I am satisfied the agent was chemical in nature, probably nitric acid, as no organisms were found and no evidence of infection ever presented itself.

Treatment consisted of: atropine sulphate 1 per cent solution daily, cold boric acid irrigations and white vaseline in the conjunctival sac. Symblepharon appeared at one small point in the left eye. This was snipped with scissors. No further complications appeared.

The prognosis given the parents was, of course, gloomy. Even considering the recuperative powers of the infant cornea the damage done was so severe as to make the outlook very poor.

The conjunctivae took on a normal appearance about July 20th, almost a month after the accident. At this time procedures for irritating the corneae were instituted, such as massage with yellow oxide of mercury ointment, instillations of dionine, and instillations of various strength solutions of glycerine in water. These procedures are used with the idea of increasing the blood flow to the anterior segment of the eye. I doubt that they had any effect on the result.

Change in the corneal opacities did not become visible until about four weeks after the accident, although the surface of the corneae became smooth before this time. Once the opacities began to clear up progress was rapid. On August 15th the right cornea was almost entirely clear, only a faint macula remaining at about 5 o'clock. The left cornea was also clear with the exception that beginning at about the center and extending toward 7 o'clock there was a fairly dense white strip containing a leash of blood vessels. The child used its eyes as any infant of its age might be expected to do. The patient was seen on November 8th, unfortunately for the last time. The opacity in the right cornea was then invisible except on illumination, and the opacity in the left cornea was decreased in size from the time of the previous examination but otherwise unchanged.

The astonishing manner in which this severe corneal damage was repaired led to some experiments in the laboratories of the Medical College of Virginia. The idea involved was to increase the nutrition of the corneal tissue and at the same time hasten the carrying away of debris by the injection of biological preparations. The results were entirely negative as regards the main object of the work, but there were some side results that might be of interest.

The first step was the production of a corneal scar in a laboratory animal that was known to be mature. Guinea pigs were used, not because they were the animals promising the best results, but because they were available in sufficient numbers and had eyes large enough for the technic involved. Silver nitrate in 10 per cent strength was used first but the resulting scar was negligible, so pure phenol

was substituted. Even this had to be used without washing and had to be repeated before a scar of suitable density was obtained. Six pigs were so treated, the left eye being used as a control. Subconjunctival injections were then made at two-day intervals, the solutions employed being: Guinea pig serum and guinea pig plasma each diluted 50 per cent with Tyrode's solution; physiological saline; and Tyrode's solution.

In looking through any work on ophthalmic therapy it will be found that subconjunctival injections are recommended for almost every diseased condition of the eye-ball. In the work just reported, they were used in an attempt to influence the clearing up of corneal scars. In this series of animals the injections were made, perforce, at a point not more than two or three millimeters from the corneal margin, as the guinea pig has a small eye. In five of these animals the injected eye fared worse than the control; in one animal it fared no better. It was considered possible that the fluid supply of the cornea was being interfered with because of injury to the conjunctiva at a point too close to the corneal margin. A second series of ten animals was prepared in the same manner, using pure phenol, and in these the injections were made under Tenon's capsule instead of subconjunctivally. The results in this second series were inconclusive, the injected eyes faring neither better nor worse than the control eyes. Judging by these results, the thought occurred that perhaps subconjunctival injections did not have as great a therapeutic value as might be assumed from reading books on ophthalmic therapy.

SUMMARY

1. Severe burn of the corneae in a new-born child, supposedly by silver nitrate.

2. Practically complete recovery of the right cornea in two months; less recovery in the left cornea, but still with a large area of clear corneal tissue.

3. Failure to aid recovery by subconjunctival injections of biological preparations in guinea pigs.

4. Possible adverse effect of subconjunctival injections as a therapeutic measure.

603 Professional Building.

Correspondence

An Improved Test for Detecting Monocular Malingering.

TO THE EDITOR:

For many years it has been known that when looking through a red glass at letters of a red hue, the letters would diminish in intensity, making them more difficult to distinguish, that is provided the opposite eye were closed.

Realizing the above fact, I did some experimental work with Mr. W. H. Delaney, and after many months, we have perfected a Malingering Test by a special printing process, whereby the red letters completely disappear. I emphasize the absolute disappearance of the letters under observation, as it is obvious that the test be an acid one that will stand up under cross fire when we render a legal opinion as to whether one is a Malingner or not.

Not only is this test worked out on the basis to determine whether the one to be examined is Malingering or not, but it is also arranged according to the Snellen Test Type, so that the amount of vision can be determined.

After the completion of this chart and finding that it served my purpose better than any other Malingering Test that we have, and after demonstrating same to our Norfolk Opthalmologists, it was accepted so favorably by those who had seen and used it, that I have given Mr. Delaney permission to copyright the chart and place it at the disposal of the Profession. A. A. BURKE, M. D.

Norfolk, Virginia.
April 10, 1935.

Analyses and Selections

The Physician Versus Proprietary and Official Preparations.

Nat Kanner, M. D., Brooklyn, in *The Virginia Pharmacist*, says:

Almost every minute of the day, some one is playing fast and loose with us. We are being hoodwinked by chicanery, and, being like birds of prey, we do not sing. The physician, intoxicated with pride and with a heart buried in the task of money-getting, has succumbed to the lure of despotic power. He has yielded to the proprietary manufacturer.

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Proprietary preparations are divided into secret and non-secret ones, of which there is a sharp distinction. Secrecy implies either fraud or avarice, usually both; of necessity it involves ignorance, and ignorance is the antipode of science. Secret medicines should *never* be employed.

A medicine of known composition, made under patent protection or otherwise controlled by a single manufacturer, may be prescribed, provided (a) that it has been honestly made; (b) that it is honestly marketed, and (c) that it is actually superior for the purpose desired to any uncontrolled preparation available. Such superiority may be either therapeutic or pharmaceutic. For the fact of its existence, however, other evidence than mere advertising claims should be required.

The physician should write prescriptions adapted to present time and individual conditions; hence he should not ordinarily order *ready-made mixtures;* and, with but few exceptions, this is equally true whether the latter be put forth as proprietary articles or be made in accordance with an official or semi-official public formula. Specification of the brand of a manufacturer, or direction of the patient to a particular pharmacist is undesirable, except when such action is necessary to assure the purity, quality or activity of the preparation dispensed. It is then perfectly proper.

Physicians have universally failed to heed the warning that they should not prescribe patent medicines, or write their prescriptions for proprietaries. It is in part, this morbid practice which has placed them in the present picture of despair, in the appalling darkness of Gethsemane.

The proprietary manufacturers, with their ever ready schemes of exploiting the profession, seem to have hypnotized us, into accepting their claims for their products, and we, like gullible targets have been their easy mark. They set the trap, and we fell for it. This would not be so dangerous a risk in itself were it not for the fact that they have attained our good-will, and through the physician's efforts, their products have been used, they then forsook the physician and advertised their products directly to the public. The result then was quite obvious, namely, *self-medication* via the patent medicine route. This is why people medicate themselves more, and see the doctor less.

Is it not time for the physician to awaken to the fact that he is constantly being exploited by the proprietary manufacturer? Doesn't the physician realize that there is no reward for him for bolstering a proprietary, only to learn later that that product has been advertised directly to the public via the newspaper, magazines, radio or drug window displays? Doesn't he know that the manufacturer has waxed rich by the use of such phrases in his advertising as, "ask your Doctor," "your Doctor recommends it," "prescribed by all Doctors," etc. He is constantly being exploited at his own expense and ultimately at the expense of the pharmacist.

Then why doesn't he get wise to himself and stop prescribing all patents, and start using again the old reliable official U. S. P. and N. F. preparations. In less than an hour he can brush up on these preparations by reviewing his textbook on pharmacology. If this could only be conveyed to all Doctors, it would be a haven of refuge to all pharmacists, it would assassinate the patent medicine kings, it would stamp out self-medication, it would end medical exploitation, and once again enrich both pharmacist and doctor alike.

So long as medical and pharmaceutical men tacitly encourage the present trend, so long will serious inroads continue to be made into private medical and pharmaceutical practice. When more physicians shall use U. S. P. and N. F. products only then will there be a material falling off in patent medicines.

Let us start right now, by a system of education, to acquaint the physician with this situation. If he learns to see the light, and learns this quickly, then you will know that it is the beginning of the end for the patent medicine.

Miscellaneous

The Tariff of Fees

Is always of interest to the profession. The tariff adopted by the Richmond Medical Profession in January, 1867, and re-affirmed January, 1887, which recently came to our hands, is given below as our readers may wish to know how they compare with fees of the present time:

MEDICAL

Each visit during the day	$ 2.00
If several patients, for each prescription	2.00

For each hour of detention after the first	$ 2.00		
Each day visit at an hour appointed by the patient or his friends	2.00	to	$ 5.00
Prescription or advice anywhere	2.00		
Night visit	5.00		
Detention all night	10.00	to	20.00
Consultation visit	10.00		
Single visit or advice (in transient cases)	5.00	to	10.00
Oral opinion of health	5.00	to	10.00
Written opinion of health	10.00	to	40.00
Stethoscopic examination (special cases)	10.00	to	20.00

OBSTETRICAL

Attendance in natural labor, including 8 visits	$40.00		
Attendance in complicated labor	50.00	to	$100.00
Abortion, exclusive of visits	20.00		
Delivery of retained Placenta	10.00	to	40.00

SURGICAL

Vaccination	$ 5.00		
Every subsequent case	2.00		
Introduction of Pessary	10.00	to	$ 20.00
Applying truss	10.00	to	20.00
Introduction speculum vaginae, first time	10.00		
Subsequent introductions, each	2.00	to	5.00
Dressing recent wounds	5.00	to	10.00
Opening abscess	2.00	to	10.00
Introduction of Catheter, first time	5.00	to	50.00
Each subsequent time	2.00	to	5.00
Introduction of Bougie, to explore	10.00	to	20.00
Hypodermic injection	2.00	to	5.00
Induction Anaesthesia	5.00	to	20.00

Public Health Statistics

I. C. RIGGIN, M. D.
Health Commissioner of Virginia

The communicable disease report received at the office of Dr. G. F. McGinnes, State Epidemiologist, during April, 1935, showed the following cases compared with the same month in 1934:

	1935	*1934*
Typhoid and Paratyphoid Fever	17	19
Diphtheria	52	65
Scarlet Fever	153	113
Measles	2,924	5,764
Meningitis	29	10
Poliomyelitis	0	0
Rocky Mountain Spotted Fever	0	0
Typhus Fever	0	0
Undulant Fever	1	2
Tularaemia	3	0
Smallpox	2	0

With the beginning of summer and the vacation period, we also come to the season of highest incidence of typhoid fever. It is at this time that many families begin to make their vacation plans. Trips to the mountains, seashore, camps, and other resorts and visits to relatives are all considered. Since these vacations necessitate travel into another locality some consult their physician regarding their safety. They ask numerous questions—"Is it safe to use milk away from home?", "Is there a good doctor at such and such a place?"—"Should we be vaccinated for typhoid?" However, the number taking these precautions are few. The majority never stop to consider these important facts concerning travel. This is particularly true of those living in the larger cities and towns where the water supply, milk, etc., are taken for granted. It is assumed that the local health authorities assume the responsibility for their safety. Nor do they consider the fact that in their travels they will possibly visit places where the sanitation is of a low order, that they may drink polluted water and unsafe raw milk. It is for this group that vaccination is urged. They should be advised that immunization for typhoid is a simple and safe procedure of proven value and one that is well worth the effort.

In 1934 the lowest morbidity and mortality rate on record in Virginia was achieved. Since the organization of the health department this decline has been gradual, but steady, due largely to the systematic and constant efforts to improve the general sanitation throughout the state, by isolation of cases and contacts, the inspection of water and sewage disposal, and the proper handling of food and milk. In 1920 there were 1,880 cases and 260 deaths from typhoid. This means that in 1920, 11.2 persons in every 100,000 died from the disease. In 1934 there were less than half as many cases, 780, and only 90 deaths. That is, the death rate is one-third

of what it was fourteen years ago. However, in recognizing how much has been done to control typhoid, care should be taken to continue these efforts, lest what has been accomplished by constant and systematic preventive work be lost and the disease become widespread again.

Typhoid vaccination cannot be advocated too strongly, particularly for those who travel, children going to summer camps, and those living in districts where the disease is endemic. We must exert all possible care to prevent further spread from primary cases, by isolation, nursing care and vaccination of contacts. With careful application of these few principles of preventive medicine a still lower typhoid rate may be expected. We ask the cooperation of the physicians of the State in carrying out these procedures.

Woman's Auxiliary to the Medical Society of Va.

President—MRS. JOSEPH BEAR, 3012 Monument Ave., Richmond.
President-Elect—MRS. FLETCHER J. WRIGHT, Petersburg.
Recording Secretary—MRS. JAMES B. STONE, 2042 Park Ave., Richmond.
Corresponding Secretary—MRS. AMBROSE MCGEE, 4107 Kensington Ave., Richmond.
Treasurer—MRS. REUBEN F. SIMMS, 2034 Park Ave., Richmond.

Auxiliary to Mid-Tidewater Medical Society.

The last meeting of this Auxiliary was held at the home of Mrs. Horace Hoskins in Saluda, April 23rd at 11:00 A. M. Plans for raising funds to continue the philanthropic work of the Auxiliary were discussed and other matters of importance were taken up at this time. At one o'clock a luncheon was held at Blakey's Hotel with Mrs. Hawes Campbell as hostess.

The meeting was resumed at two-thirty o'clock at the home of Mrs. Hoskins with an attractive program. Mrs. R. D. Bates read an interesting account of Colonial Medicine. Mrs. Horace Hoskins gave the poem "To Physicians Everywhere." A paper on the "Life of Jane Todd Crawford" was read by Mrs. Hawes Campbell. The program was well received by a large attendance.

This auxiliary has made splendid progress under the leadership of Mrs. Hawes Campbell, of Enfield. The membership has steadily increased and a great deal of interest has been manifested at all meetings. It furnishes a "Loan Closet" consisting of linen supplies, adult and infants clothes and many articles necessary to the sick, which the doctors of King William County have found of great service in the care of indigent patients.

The Woman's Auxiliary to the Norfolk County Medical Society

Held a card party and dance on February 15th. Approximately 600 people attended. In the afternoon there were fifty tables of bridge while at night there were about seventy. After the card playing there was a dance. Everyone had a most enjoyable time and the receipts were over $310 which will be used for the philanthropic work of the organization.

It is planned to have a committee composed of Mrs. Southgate Leigh, Mrs. M. N. King, Mrs. R. L. Peake and Mrs. C. C. Smith to decide what use shall be made of the proceeds.

On April 15th the Auxiliary participated in a Public Relations tea with the Woman's Club, the speakers being two of the Norfolk doctors. This was very well attended.

The Richmond Auxiliary

Held its annual card party on May 15th at the home of Dr. and Mrs. William T. Sanger in Hillcrest. It was a lovely party and well attended. Considerably more than $100 was realized.

Through the kindness of local druggists, very nice prizes were donated. Door prizes were also given by friends.

The funds in hand will be applied to the welfare work done by the Auxiliary. Recently $75.00 was given the Out-patient Department of the Medical College of Virginia for glasses and for the dental care of indigent patients. The milk fund for undernourished school children is still being provided.

Extract from a National News-Letter.

The Florida Medical Advisory Board and the State Medical Auxiliary Board held a joint meeting recently to formulate some definite work for the coming year. After these plans had been approved by the State Medical Association they were sent out to the various county groups throughout the state. The Woman's Medical Auxiliary of Duval County has adopted an excellent program with seven di-

visions, each covering an entirely different phase of work. They are as follows:

"1. Let each member at all times hold herself in readiness to serve on health committees of any outside organization.

"2. Assist in the dissemination of good health literature by means of an enlarged circulation of *Hygeia.*

"3. Give your wholehearted co-operation to the program of Cancer Prevention and arrange, if possible, one public presentation of Cancer Prevention material in your county, several if advisable, at a time acceptable to the state program.

"4. Assist in the organization of new county auxiliaries.

"5. Hold yourself in readiness to give as wide support as possible when called upon by the State Legislative Committee.

"6. Present to as many organizations as possible the talk on 'The Necessity of Health Cards for Household Servants.'"

(NOTE—This talk deals with the necessity for requiring household servants the same health certificate that is at present required of food handlers. This is advocated for the purpose of lessening the danger from venereal disease as well as tuberculosis.)

"7. Use at least one study envelope during the year. These are for your information and will prove of much value to you if you will choose one or more and assign topics to different members for presentation."

Truth About Medicine

In addition to the articles previously enumerated, the following have been accepted by the Council on Pharmacy and Chemistry of the A. M. A.:

Wm. S. Merrell Company

Diphtheria Toxoid, Alum Precipitated (Refined), ten 1 c.c. vials (10 immunizations) package.

U. S. Standard Products Co.

Polyanaerobic Antitoxin (Tetanus-Gas-Gangrene), Refined and Concentrated.

PROPAGANDA FOR REFORM

Pertussis Vaccine, Immunizing (Sauer), (P. D. & Co.)—In a report on pertussis vaccine prepared after the method of Dr. Louis W. Sauer and marketed by Eli Lilly & Co. (The Journal, March 3, 1934, p. 692), it was stated that the Council does not feel justified at this time to recognize the use of pertussis vaccine of any sort for therapy or prophylaxis until more convincing evidence becomes available. Recently Parke, Davis & Co. presented an identical preparation for consideration of the Council. In accordance with its previous decision, the Council voted to consider this preparation as having the same status as that manufactured by Eli Lilly & Co. and postponed consideration to await the evidence to determine its value. (J. A. M. A., March 9, 1935, p. 834.)

Cod Liver Oil.—Cod liver oil has long held a prominent place among therapeutic agents. Despite studies that have revealed the enormously greater antirachitic potency of viosterol as gaged by the conventional bioassay methods, cod liver oil is still highly regarded. There is evidence that, based on units of vitamin D as determined by assay on rats, cod liver oil is superior to viosterol in the cure and prevention of rickets in human infants. Despite the fact that nearly all fish oils contain vitamin D, perhaps the most striking support for the peculiar efficacy of cod liver oil has been recently brought forward by Bills, Massengale and Imboden, who found that blue fin tuna liver oil with 40,000 units of vitamin D per gram was only one-sixth as effective when assayed with chicks as was cod liver oil with only 100 units of vitamin D per gram. These and other observations have suggested that there are several forms of vitamin D. Holmes and Remington have shown that American cod liver oil contains more iodine than do most ordinary foods (calculated to the dry basis). The content of iodine varies with the place where the fish are caught, the average of the values being nearly nine parts per million. However, it is not to be concluded that cod liver oil should be administered for its iodine content. (J. A. M. A., March 16, 1935, p. 926.)

Aspirin-Bayer.—The original home of Aspirin-Bayer was in Germany. Neither that country nor any others of importance would grant a patent either on the product acetylsalicylic acid or on the process of making it. The United States patent office granted both. As a result, for seventeen years it was impossible for any one in our country to manufacture or sell acetylsalicylic acid except the Bayer Company. This monopoly made it possible for the company to exploit the American medical profession and the public by charging an exorbitant price for aspirin. In February, 1917, the patents on aspirin expired. Any pharmaceutical house may now manufacture acetylsalicylic acid and sell it under the name aspirin. There followed a period during which the Bayer concern carried on an advertising campaign which led the public to believe that the only "genuine aspirin" on the American market was the Bayer product. The same campaign also stressed the alleged harmlessness of Aspirin-Bayer. In September, 1934, the Federal Trade Commission ordered the company to cease and desist from representing in newspaper and magazine advertising or over the radio that aspirin is the trademark of the Bayer brand of acetylsalicylic acid and from making such claims as "It does not depress the

heart," "It cannot harm the heart," "Bayer aspirin is always safe," "You could take it every day without harm," and many other similarly false and misleading claims. The retail price of Aspirin-Bayer has been brought down to a price more nearly comparable with competing brands. (J. A. M. A., March 23, 1935, p. 1009.)

Book Announcements

Recent Acquisitions to the Library, Medical College of Virginia.

The following and other books in the Library of the Medical College of Virginia may be borrowed by members of the Medical Society of Virginia, the only charge being return postage:

American Dental Association—Accepted Dental Remedies.
Bainton & Burstein—Illustrative Electrocardiography.
Barborka, C. J.—Treatment by Diet.
Beard, C.—Whither Mankind?
Beck, B. F.—Bee Venom Therapy.
Bennett, M. E.—College and Life.
Black and Davis—New Practical Physics.
Booklet describing the Leper Settlement at Sungei Buloh.
Briggs, T.—Secondary Education.
Burn, J. H.—Method of Biologic Assay.
Charters & Waples—The Commonwealth Teacher Training Study.
Chevalley, A. and M.—The Concise Oxford French Dictionary.
Commonwealth Fund—A Study of Rural Public Health.
Copeland, R. S.—Home Medical Book.
Corsi, E.—In the Shadow of Liberty.
Counts, G.—American Road to Culture.
Cowdry, E. V.—A Textbook of Histology.
Crile, G.—Diseases Peculiar to Civilized Man.
Curriculum for Schools of Nursing.
Ditmars, R. L.—Confessions of a Scientist.
Doyle—Nursing by Religious Orders in the U. S.
Elmer and Rose—Physical Diagnosis.
Flugel, J. C.—A Hundred Years of Psychology, 1833-1933.
Földes, E.—A New Approach to Dietetic Therapy in Epilepsy.
Foote, J. A.—State Board Questions and Answers for Nurses.
Goldzieher, M. A.—Practical Endocrinology.
Haire, N., ed.—Encyclopedia of Sexual Knowledge.
Hamilton, A.—Industrial Toxicology.
Harap, H.—Economic Life and the Curriculum.
Henderson, Y.—A New Deal in Liquor.
Hess, Mohr and Bartelme—The Physical and Mental Growth of Prematurely Born Children.
Hopkins, L. T.—Curriculum Principles and Practices.
Hunt, J. R., et als.—The Biology of the Individual.
Johns & Pfefferkorn—An Activity Analysis of Nursing.
Kenealy, A.—The Human Gyroscope.
Kretchmer, E.—A Textbook of Medical Psychology.
Lea and Febiger—One Hundred and Fifty Years of Publishing.
Lee, E., and others—Objectives and Problems of Vocational Education.
Leschke, E.—Clinical Toxicology.
Lowell—At War with Academic Traditions in America.
Macleod—Physiology. New ed.
Malamud, Wm.—Outlines of General Psychopathology.
Meigs, J. V.—Tumors of the Female Pelvic Organs.
Morris, R. T.—Fifty Years a Surgeon.
Moyer and Fittz—Air Conditioning.
Munson & Stevens—Story of the National League of Nursing Education.
Musser, J. H.—Internal Medicine: Its Theory and Practice.
Needham, J.—A History of Embryology.
Nursing Schools Today and Tomorrow.
Ogino, K.—Conception Period of Women.
Osborn, F. A.—Physics of the Home.
Osborne, O. T.—Mouth Infection.
Pardee, H. E. B.—Clinical Aspects of the Electrocardiogram.
Pope & Young—The Art and Principles of Nursing.
Prinz & Greenbaum—Diseases of the Mouth.
Purves-Stewart—Diagnosis of Nervous Diseases.
Rivers, T. M.—The Autonomic Diseases or Rheumatic Syndrome.
Seabury, D.—What Makes Us Seem So Queer?
Semon, H. C. G.—An Atlas of the Commoner Skin Diseases.
Sherman, H. C.—Food and Health.
Squire, A. O.—Sing Sing Doctor.
Study on the Use of the Graduate Nurse for Bedside Nursing in the Hospital.
Symington, J.—In a Bengal Jungle.
Thoma, K. H.—Clinical Pathology of the Jaws.
Walsh, W. S.—Making Our Minds Behave.
Waples & Tyler—Research Methods and Teachers Problems.
Waters & Kaplan—Yearbook of Radiology, 1934.
Wheeler, J. B.—Memoirs of a Small-Town Surgeon.
Wheelwright, E. G.—The Physick Garden.
Whitehead, A. N.—Aims of Education and Other Essays.
Wiggers—Physiology in Health and Disease. New ed.
Zinsser, Hans—Rats, Lice and History.

Diseases of the Skin. By RICHARD L. SUTTON, M. D., Sc. D., LL. D., F. R. S. (Edin.), Professor of Dermatology, University of Kansas, School of Medicine. And RICHARD L. SUTTON, JR., A. M., M. D., L. R. C. P. (Edin.), Assistant in Dermatology, University of Kansas, School of Medicine. Ninth Edition. Revised and Enlarged. St. Louis. The C. V. Mosby Company. 1935. Octavo of 1,433

pages. With 1,310 illustrations and 11 colored plates. Cloth. Price, $12.50.

Every practitioner needs in his library a standard work on dermatology. When it attempts to fill many needs by additions in successive editions, it grows and grows until it becomes almost encyclopaedic. This is true of the ninth edition of Sutton's work just published.

Medical authors, who submit their books to colleges for students use should be compelled to come and meet the men, for after all, a book is a message, and the message becomes vital with personal acquaintance. The reviewer, having this privilege, feels the background of this book quite as interesting as the text.

A most able physician elects dermatology as his field of work, and after several years gives to the world, the result of his observations and experience. He does this fully using previous older books as models, but changing and adding his personality to the whole. The result is Richard Sutton's Diseases of the Skin, published in 1916 and a most satisfactory performance.

Now, this man has a son coming on in the same field. The new and modern appeals to him and into the book, the new must come, but always there is that governing mind of great clinical judgment to say just how far he may go.

This situation produces, as would be expected, more than a one man result, but there is a complete unity in scheme and presentation. To the specialist, this book is particularly desirable because of the bibliography which is appended to each subject. It is not a book for students. This need is met by their "Introduction to Dermatology" but it is a book to which every student should have access.

Its field of usefulness is tremendously wide. Its illustration and the idealism of the publisher, as expressed in the technique of printing, make it a very desirable book.

THOS. W. MURRELL.

Contribution of Statistics to the Prevention of Blindness; the Control of School Myopia; Eye Health for Atypical Children; Community Enterprise in Preventing Blindness; the Philosophy Underlying the Education of the Visually Handicapped; the Sight-Saving Class as a Mental Hygiene Measure; the Broad Basis for Prevention of Blindness; Preventing Blindness Throughout the World; Leisure Time Activities for Sight-Saving Class Pupils; Trachoma in Wrestlers: A Menace to Public Health; Corrective and Protective Eye Goggles for Coal Mine Employees. Pamphlets published by the National Society for the Prevention of Blindness, Inc. 50 West 50th St., New York, N. Y. Ranging in price from 5 to 20 cents.

Hospital Service in the United States. 1935. Fourteenth Presentation of Hospital Statistics by the Council on Medical Education and Hospitals of the American Medical Association. The 1934 Census of Hospitals. Hospitals Registered by the American Medical Association. Pamphlet reprinted from the Hospital Number of the Journal of the American Medical Association, March 30, 1935. Price, 50 cents.

The Modern Method of Birth Control. By THURSTON SCOTT WELTON, M. D., F. A. C. S., Editor of the American Journal of Surgery. Walter J. Black, Inc. New York. 1935. Octavo of 168 pages. Cloth. Price $3.00 net.

Birth control is one of the important controversial subjects of today. It is openly discussed everywhere. The New Method, that of Ogino and Knaus, described by the author, is scientifically correct. Whether it will work out in practice, it is of course too early to say. Dr. Thurston's guide is evidently written for the laity, and as often is the case in trying to make things simple we become abscure. To say the uterus is the size of a pear is not as clear as giving dimensions in inches and to call spermatozoa father-cells does not make for clearness. Personally I object to "safe" and "unsafe" being applied to parts of the menstrual cycle when "unfertile" and "fertile" is meant. Why the author should say that the mothers blood flows through the cord to the child, I do not see.

There is an excellent Summary in Chapter VII that gives one the contents of the book in a clear and concise manner.

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Editorial

The Safe Period.

Since 1929 the theories of Knaus and Ogino concerning periodic fertility and sterility in women have occasioned increasing attention and discussion among the profession, even among the laity. Of the two, Hermann Knaus's voice has been the louder. In fact as one reads his book it is hard to swallow some of his Teutonic braggadocio and conceit. Perhaps in one who takes himself so seriously such an attitude is to be expected. At all events his theories may now be regarded as the talk of the town among those who are concerned with birth restriction and functional sterility.

It seems that in 1924 Professor Knaus undertook research work on the pharmacology and physiology of uterine muscle under Professor A. J. Clark in London, and that the next year he continued his work at Cambridge. His investigations led to the study of the corpus luteum as a gland of internal secretion and in 1927 he advanced the idea that a lutein hormone produces a typical reaction in the uterine mucosa and muscle—hypotonia, swelling and non-responsiveness to pituitrin. Having demonstrated this reaction in rabbits to his own satisfaction he found that the same rule seemed to hold for the human uterus. By means of a water filled balloon in the uterus connected with a kymograph he claims in normal women with regular periods to have fixed the time of ovulation. He found that the changes in the uterus registered by the instrument occurred sharply and always at a fixed time in the menstrual cycle. His ability to determine the moment of extrusion of the unfertilized ovum from a ripened Graafian follicle is the pivotal point in Knaus's theory, first advanced by him in 1929.

In accepting Knaus's theory of periodic fertility and sterility the following biological facts must be agreed to: (1) The life of the unfertilized ovum after ovulation is extremely brief—not longer than twenty-four hours in the human being; (2) The viability of human spermatozoa within the genital tract or abdomen of the female is not much longer—forty-eight hours at the most; (3) "The corpus luteum metoestrum in normal woman has an autonomous function regularly lasting fourteen days"; (4) Ovulation in women who menstruate regularly always normally occurs on the same relative day of each menstrual cycle—on the fifteenth day before the onset of menstruation.

If these statements are true the period of each menstrual cycle during which women are fertile and can therefore become impregnated is definite and relatively short—only three days. In practice however, for sake of additional safety, the fertile period is regarded as extending over five days, and in theory at least it becomes necessary for women desiring to avoid pregnancy to abstain from coitus only dur-

ing these few days. Knaus maintains that at any other time in the month it is impossible for them to conceive. As this rule applies only in cases of absolute regularity of menstruation such factors as illness, pregnancy, and change of climate have to be allowed for and in instances of irregular menstruation much more extreme limits have to be fixed and adhered to. In fact all of the reported failures in those who have attempted to follow Knaus's rules are explained by him on the basis of miscalculations due to faulty knowledge concerning the length and rhythm of the particular menstrual cycle. Great care is enjoined on all women who are desirous of applying Knaus's rules in their sexual life to begin by observing and recording the first day of their menstrual cycle over a period lasting at least a year, such data being essential in determining what type of menstrual cycle characterizes the particular individual. To assist in this most important part of his plan the author provides his patients with specially aranged menstrual calendars.

In substance Professor Knaus claims that a woman who menstruates regularly every twenty-eight days will ovulate on the fifteenth day before the onset of her next expected period. Assuming this to be the fifteenth of June she may regard June twelfth to the sixteenth inclusive as the only days of that month upon which coitus will be followed by conception, and if she be the average normal woman that result is practically assured if intercourse is practised during these five days of fertility.

Assuming the case of another woman who menstruates at a regular interval of twenty-six to twenty-nine days, ovulation occurs each month between the twelfth and fifteenth days of the cycle inclusive and this constitutes what has been called "the ovulation period." When the two days preceding the ovulation period are added in and an additional day allowed for at the beginning and end of the whole time we find that the fertile period (ninth to fifteenth day inclusive) in this case is extended to eight days.

If this theory of periodic fertility and sterility is true it makes unnecessary the elaborate contraptions of the contraceptive clinic and puts the control of conception upon a surer basis than now exists. By following such methods it has been suggested that not only may childbirth be controlled in a more acceptable way even to present opponents of the practice but that cases of supposed sterility may often be corrected. It still remains to be proved beyond peradventure of a doubt whether or not Professor Knaus's theory is true.

Delegate Hobson's Bill.

Some time ago the Richmond *Times-Dispatch* took occasion editorially to call our attention to Delegate Haskins Hobson's bill for the creation out of public funds of an institution in this state for the treatment of alcoholic addicts. The measure was defeated at the 1932 session of the General Assembly but the newspaper states the author is contemplating reintroducing it next year. It labels the bill "a thoroughly meritorious one" and believes it "should command the support of both wets and drys." The editorial speaks glibly of "modern corrective treatment" and of returning the patients "to their several communities morally and physically rehabilitated, fitted for the serious work of good and useful citizens." It evinces considerable horror that the state should continue to care for chronic alcoholism in "the alcoholic wards of an asylum for the insane."

In dealing with this question it is unfortunate that our lay writer does not employ the Virginia statutory title by which our state institutions are designated. We may be forgiven for reminding him that we no longer have asylums for the insane in Virginia. Each of our institutions is called a hospital—for example, there is the Central State Hospital, the Western State Hospital, etc.—and each is supposed to be conducted upon the high scientific plane that that title implies.

The second mistaken idea that seems to underlie the *Dispatch's* praise of Delegate Hobson's bill is the mediaeval conception that mental disease is something horrible, something to shun, or be ashamed of. From what we can make out the writer is afraid the "insane" may do something to the alcoholic. The impression is given that the nice sufferer from chronic alcoholism may be harmed by contact with the disreputable victim of mental disease. As a matter of fact quite the opposite is possible.

There is nothing to be ashamed of in inherited mental disease. We might be justified in showing less patience for acquired mental disease. So-called "insanity," drug addiction, alcoholic addiction, all are forms of mental disease, and the sufferer from

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each is to be pitied, not scorned, shunned and scandalized.

At present about 500 of the state sufferers from alcoholic addiction are being taken care of annually at one of our state hospitals. If they are not being properly taken care of, mere isolation will not solve the difficulty. They would still be under the supervision of a lay board. There is much room for improvement in the care of all our mental wards in the State of Virginia. The fault lies not in the medical profession but in the pitiful fact that politics and lay bodies now mold the destinies of our 10,000 mental patients.

After Fifty Years.

The eleventh annual meeting of the American Association of the History of Medicine was dedicated to the memory of an American physician of genius and self-sacrificing devotion to an idea—Joseph P. O'Dwyer. There are few of us who do not know that O'Dwyer invented an improved and perfected laryngeal intubation tube, a life saving device in laryngeal diphtheria. Before O'Dwyer's time laryngeal diphtheria exacted a fearful toll of the infant and child population. His invention brought hope and relief where formerly there had been none.

O'Dwyer was born in Cleveland, Ohio, in October, 1841. He graduated in medicine from the College of Physicians and Surgeons in New York City in 1866. He twice contracted cholera while attempting to stem the ravages of cholera epidemics. His work on laryngeal diphtheria and its treatment by intubation began with his appointment to the staff of the New York Foundling Hospital in 1872. Painstaking studies on numberless plaster casts of larynx and trachea, repeated trials and failures with tubes of various materials and various shapes in a search for one that would not be irritating, that would not slip into the trachea, that could not be expelled by coughing, and into which food and liquid could not be aspirated—by such a course he arrived at the successful invention which he described for the first time fifty years ago, and for which today we take occasion to acknowledge our indebtedness to him.

Department of Clinical and Medical Education of the Medical Society of Virginia

Postgraduate Clinics.

The Fifteenth Postgraduate Clinic was conducted at the University of Virginia Hospital in coöperation with the Department of Clinical and Medical Education of the Medical Society of Virginia, in accordance with the previously announced program. Forty-six physicians, in addition to members of the Medical School and Hospital staffs attended. They came from as distant posts as Emory in the Southwest and Newport News in the East. Hospital officials report that this was one of the most, perhaps the most, successful of the postgraduate clinics conducted at the University of Virginia. The names and addresses of the physicians who registered for the clinic are as follows:

Dr. W. C. Archer, Waynesboro.
Dr. J. B. Bailey, Keysville.
Dr. H. L. Baptist, Ivy Depot.
Dr. G. B. Barrow, Staunton.
Dr. N. Bloom, Richmond.
Dr. R. E. Booker, Lottsburg.
Dr. Glenn C. Campbell, Staunton.
Dr. C. C. Canada, Staunton.
Dr. F. T. Cassidy, Fredericksburg.
Dr. E. M. Chitwood, Wytheville.
Dr. John E. Cole, Fredericksburg.
Dr. Lee Cooke, Fredericksburg.
Dr. J. C. Coulter, Charlottesville.
Dr. H. S. Daniel, Louisa.
Dr. E. D. Davis, Jr., Crozet.
Dr. Clive O. Dearmont, White Post.
Dr. C. G. Grazier, U. S. Navy.
Dr. Percy Harris, Scottsville.
Dr. H. T. Hawkins, Waynesboro.
Dr. R. L. Hillman, Emory.
Dr. Lewis Holladay, Orange.

Dr. H. B. Holsinger, Farmville.
Dr. J. F. Hubbard, Waynesboro.
Dr. D. M. Kipps, Front Royal.
Dr. W. A. Kyger, Free Union
Dr. Anita Lotti, Charlottesville.
Dr. W. R. Martin, Charlotte C. H.
Dr. S. G. Miller, Piney River.
Dr. G. L. Morriss, Victoria.
Dr. J. O. Mundy, Charlottesville.
Dr. W. A. Murphy, Staunton.
Dr. Marshall J. Payne, Staunton.
Dr. B. A. Rice, Forest.
Dr. L. G. Roberts, Moorman's River.
Dr. Alex. F. Robertson, Jr., Staunton.
Dr. Chas. W. Scott, Newport News.
Dr. J. H. Scherer, Richmond.
Dr. Elizabeth B. Sherman, Front Royal.
Dr. J. F. Thaxton, Tye River.
Dr. W. H. Turner, Round Hill.
Dr. A. E. Turman, Richmond.
Dr. H. M. Wallace, Staunton.
Dr. W. R. Warren, Woodberry Forrest.
Dr. Chas. E. Watson, Broadway.
Dr. J. A. Wolfe, Staunton.
Dr. F. J. Wright, Petersburg.

The Christiansburg Medical Society has under way plans for a postgraduate clinic in Pediatrics which is to begin later in the year.

GEORGE B. ZEHMER, *Executive Secretary.*

Proceedings of Societies

The Postgraduate Medical Society of Southern Virginia

Met in conjunction with the Department of Clinical Education of the Medical Society of Virginia in the courthouse at Surry on the afternoon of Tuesday, May 7th. The president, Dr. H. E. Whaley, of Victoria, presided. The secretary-treasurer, Dr. C. E. Martin, of Emporia, reported all bills paid to date and a satisfactory response on the part of members relative to their financial obligations to the Society.

Several interesting case reports were submitted which were discussed by Drs. R. L. Raiford, W. W. Seward, A. S. Graham, J. E. Rawls and others.

Dr. Hyman Cantor, of Petersburg, presented a paper on "Acute Perforation of Gastric Ulcer"; Dr. R. L. Raiford, of Franklin, presented a paper on "The Use of Plaster Splints in Treating Fractures." The invited guest, Dr. A. Stephens Graham, of Richmond, gave a most instructive talk on "The Colon," considering its embryology, physiology and pathology, followed by a resumé of the various neoplastic lesions commonly found in various parts of the colon with an outline of modern methods of diagnosis and treatment.

At the business session which followed the scientific program, Dr. A. T. Brickhouse, of Hopewell, was elected to membership. Dr. F. E. Steere, of Claremont, was elected delegate and Dr. W. W. Seward, of Surry, alternate to represent the county of Surry at the Norfolk meeting of the Medical Society of Virginia.

The next meeting of the Postgraduate Society will be held in Emporia, at 2:30 P. M., Tuesday, August 27th.

The Mid-Tidewater Medical Society

Met at Saluda, Va., on April 23rd, for its regular quarterly meeting with about thirty members and guests in attendance. Dr. E. L. W. Ferry, of Millers Tavern, presided and Dr. M. H. Harris, of West Point, was at the secretary's desk.

Several matters of business were attended to at the morning session, and the following resolution was adopted:

WHEREAS, It has been brought to the attention of the Mid-Tidewater Medical Society, composed of the counties of Essex, Middlesex, Gloucester, Mathews, York, New Kent, King William and King and Queen, that the clinics at the Medical College of Virginia and at the University of Virginia are treating many cases that are not indigent;

BE IT RESOLVED, That the Mid-Tidewater Medical Society asks the cooperation of these two clinics in checking this unfair and uncalled for competition, and that a requirement for treatment shall be a written request from the family doctor, before receiving treatment;

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FURTHER BE IT RESOLVED, That a copy of this resolution be sent to the head of each clinic and a copy be reported to the Medical Society of Virginia publication.

Between the morning and afternoon sessions, a delicious dinner was served by the physicians of the county of Middlesex to the members and guests.

At the afternoon session papers were given by Dr. Fred M. Hodges, of Richmond, on "Treatment of Cancer with X-ray and Radium." This was illustrated with slides, showing the improvement and benefit, with disfigurement. Dr. Guy W. Horsley, also of Richmond, read a paper on "Report of Five Hundred and Two Cases of Appendicitis at St. Elizabeth's Hospital between January 1, 1931 and December 31, 1933." Both papers were discussed and many questions were answered.

The next meeting will be held at Gloucester in July.

The Virginia Society of Oto-Laryngology and Ophthalmology

Held its sixteenth annual meeting at the Stratford Hotel, Fredericksburg, Va., on May 4th. The president, Dr. Beverley R. Kennon presided. The mayor of Fredericksburg, Dr. R. J. Payne, welcomed the members to the city. There were thirty-nine in attendance. Among the invited guests were Drs. Ben Witt Key and M. C. Myerson, of New York, who presented interesting papers. Other prominent men on the program were Drs. H. S. Hedges, Fletcher D. Woodward, E. T. Gatewood, M. H. Hood, F. H. McGovern, and Orvin C. Jones.

The Society decided to sponsor another Postgraduate Course at the University of Virginia this year.

The following officers were elected for the ensuing year: President, Dr. Emory Hill, Richmond; vice-president, Dr. J. Minor Holloway, Fredericksburg, and secretary-treasurer, Dr. George G. Hankins (re-elected), Newport News. The next meeting will be held in Richmond, Va., in May, 1936.

Nansemond County Medical Society.

At the March meeting of this Society Dr. E. C. Joyner was re-elected president for the ensuing year and Dr. William F. Wild was elected secretary. Both are of Suffolk.

The Society was addressed by Dr. J. Shelton Horsley, of Richmond, at its meeting on April 6th, his subject being "Cancer of the Stomach and Intestines."

Dr. William B. Porter, of the Department of Medicine, Medical College of Virginia, spoke at the meeting on May 9th.

The Amelia County Medical Society

Held a regular meeting at Amelia C. H. on May 6th, at which time the following officers were elected for the ensuing year: Dr. James L. Hamner, Mannboro, president; Dr. G. C. Eggleston, Amelia, vice-president; and Dr. James L. Hamner, secretary-treasurer.

Dr. J. M. Habel and Dr. H. C. Rucker were elected delegate and alternate, respectively, to the State meeting to be held in Norfolk this fall. Upon the request of Governor Peery, Dr. J. M. Habel and Dr. J. L. Hamner were selected as delegates to the Statewide Safety Conference to be held in Richmond, May 15th and 16th.

The South Piedmont Medical Society

Held its semi-annual meeting at Danville, Va., April 23rd, under the presidency of Dr. George A. Stover, South Boston. There was a symposium on "Medical Economics" in addition to several papers on other subjects, and the program was most interesting.

The following officers were elected for the ensuing year: President, Dr. J. J. Neal, Danville; vice-presidents, Dr. H. R. Bourne, Danville; Dr. George T. Harris, Lynchburg; Dr. C. B. White, Halifax; Dr. H. C. Chalmers, Phenix; and secretary-treasurer, Dr. J. A. Owen (re-elected), Turbeville. The next meeting will be held in South Boston on Tuesday, November 26, 1935.

The Southampton County Medical Association

Met in the office of Dr. E. A. de Bordnave, Franklin, Va., on the afternoon of April 11th. The meeting was called to order by Dr. John N. Applewhite, president. In the absence of Dr. W. T. McLemore, Dr. R. L. Raiford acted as secretary *pro tem.* There was no business to be considered and the reading of the minutes of the last meeting was dispensed with.

Dr. Ambrose McGee, of Richmond, addressed the meeting on "Some of the Feeding Problems of Children," after which a general round table discussion was entered into. There was much interest

shown and all present expressed the opinion that this was one of the best meetings of the Society.

The Roanoke Academy of Medicine

Held its fourth hospital meeting at the Roanoke Hospital, Monday evening, April 29th, at which time the following scientific program was presented:

1. Carcinoma of Thyroid—Dr. L. D. Keyser.
2. Torsion of Omentum—Dr. T. D. Armistead.
3. Glycocoll Therapy in Muscular Dystrophy—Dr. L. Davis.
4. Continuous Irrigation in Peritonitis—Dr. J. P. Kent and Dr. C. M. Irvin.
5. Carcinoma of Penis—Dr. S. M. Novak and Dr. T. D. Armistead.
6. Poly-Orchidism with Malignancy in the Supernumerary Testicle—Dr. W. L. Powell and Dr. L. D. Keyser.

Dr. W. P. Jackson and Dr. Frank Helvestine, Jr., both of Roanoke, are president and secretary, respectively.

The Physicians Journal Club of the Eastern Shore of Virginia

Held its regular meeting at Accomac, Va., on May 14th. At this time, interesting papers were presented by Dr. Southgate Leigh, Jr., Norfolk, on Minor Surgery, and by Dr. Ira Hurst, Parksley, on The Treatment of Diabetes by the High Carbohydrate-Low Fat Diet.

Though the two counties on "The Shore" have independent organizations, the Journal Club has been an excellent means for getting together the profession of that section and for promoting a splendid feeling of good will between the doctors of the two counties.

News Notes

American Medical Association.

The 86th annual session of the Association is to be held at Atlantic City, N. J., June 10th-14th. Dr. Walter L. Bierring, of Des Moines, Iowa, is president and Dr. James S. McLester, of Birmingham, Ala., will succeed to the presidency at this time. This session gives promise of being one of the most interesting and pleasant in the history of the organization as for the first time in its history there is to be a joint session with Canadian Medical Association.

The House of Delegates will convene on Monday, the 10th, at 10:00 A. M., and the Scientific Assembly will open with the General meeting on Tuesday, the 11th, at 8:30 P. M. Several entertainments and group dinners will add much to the social interest of this session.

Norfolk Meeting of State Society.

The local committee in charge of arrangements for the Norfolk meeting of the Medical Society of Virginia has been working on details for the meeting to be held in that city on October 15th, 16th and 17th. The Monticello will be hotel headquarters and all sessions and exhibits will be held there. As far as possible, members should make early reservations to insure securing accommodations in the hotel of their choice. This session promises to keep up the reputation made by Norfolk as a fine meeting place.

The Program Committee of the Society hopes to have an attractive program for announcement at an early date.

State Child Conservation Committee Formed.

Upon the invitation of Dr. W. P. Jackson, Chairman of the Child Welfare Committee of the Medical Society of Virginia, and Miss Ellen H. Smith, Chairman of the Child Hygiene Committee of the Cooperative Education Association, Virginia Parents and Teachers, a number of child hygiene and child welfare committees of Statewide Associations met in Richmond on April 3rd to coordinate their efforts, forming the *State Child Conservation Committee*. The organizations and associations represented in this group are: The Medical Society of Virginia, with Dr. W. P. Jackson as chairman; the Virginia Tuberculosis Association, with Dr. Dean B. Cole as Chairman; the Virginia Pediatric Society, with Dr. J. B. Stone as chairman; the Cooperative Education Association, Virginia Parents and Teachers, with Miss Ellen H. Smith as chairman; the Virginia Education Association, with Mr. J. M. Shue as chairman; the State Superintendents

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Association, with Mr. W. A. Scarborough as chairman.

Virginia's status among the states of the nation, and particularly the Southern States, was considered by this group and it was concluded that, in health measures, affecting particularly the welfare of mothers and children as well as the protection of all citizens, Virginia was not maintaining her place even among the Southern States.

Miss Smith indicated the five outstanding points which needed the attention of the group as:

1. Correction of certain remediable physical defects of indigent school and pre-school children: vision, tonsils, adenoids, teeth, and immunization for them.
2. Better facilities for the early diagnosis and early treatment of tuberculosis.
3. Better obstetrical service.
4. Venereal disease control.
5. Provision for a continuous postgraduate service to Virginia practicing physicians.

Committees were appointed to consider these recommendations and to report to the State Child Conservation Committee at a meeting in Richmond on June 1st. Indications are that requests for necessary funds to support these measures will be carried to the Legislature.

New Health Officers Appointed.

Dr. J. B. Porterfield has been appointed health officer of Montgomery County with headquarters at Christiansburg, Va.,-the appointment having become effective May 1st. He is a graduate of the University of Virginia, class of '33, and interned at the Toledo Hospital, Toledo, Ohio.

Dr. Linwood Farley, who has been practicing for some time at Courtland, Va., has been assigned to the Southwest Health District as assistant to Dr. Edgar C. Harper, deputy director of rural health in that area, with headquarters at Abingdon. Dr. Farley's appointment became effective May 1st.

Dr. George E. Waters, of the class of '33, University of Virginia, who later interned at the Baltimore City Hospital, has been appointed acting director of the Peninsula Health District, effective May 6th. He succeeds Dr. C. H. Dawson resigned. This district is composed of the counties of James City, Elizabeth City, Warwick and York, with headquarters at Williamsburg.

All of the new health officers named above recently completed courses in Public Health at Johns Hopkins University and have been engaged in health work in Maryland.

The Virginia Academy of Science

Held its annual meeting at the University of Richmond early in May with an attendance of over 400. The sessions were both interesting and instructive. The Academy's Committee on research, headed by Dr. J. Shelton Horsley, announced that Miss Margaret Hess, University of Virginia graduate, won the Academy's prize for the best paper presented during the year. This was based on research done in the department of biology at the University. Research grants were awarded the following: Dr. J. W. Beams, of the University of Virginia; W. B. Bell, Virginia Polytechnic Institute; Dr. Walter S. Flory, Bridgewater College; Dr. H. B. Haag, Medical College of Virginia; Dr. C. C. Speidel, University of Virginia; and Dr. Edward Steidtmann, of the Virginia Military Institute.

Dr. Ida Sitler, head of the Hollins College science department, took office as president for the coming year, succeeding Dr. W. T. Sanger, president of the Medical College of Virginia. Dr. H. E. Jordan, of the University of Virginia, was named president-elect, and Dr. E. C. L. Miller, Medical College of Virginia, was re-elected secretary-treasurer. The 1936 meeting will be held at the Virginia Military Institute, Lexington, Va.

University of Virginia Hospital, School of Nursing,

Held its Commencement Exercises in Madison Hall, at the University on May 9th. The graduates, who were awarded their diplomas by President John L. Newcomb, were presented by Dr. J. Carroll Flippin, Dean of the School of Medicine. Following the presentation of University Hospital Alumnae Pins by Dr. John H. Neff, the graduates were addressed by Dr. Frank A. Geldard.

After the ceremonies a tea was tendered the graduates who numbered fifty-eight including ten transfers from the Mary Washington Hospital, Fredericksburg, eleven from Sheltering Arms Hospital, Richmond, four one-year affiliates each from Blue Ridge Sanatorium and from Catawba. The Mary Washington and the Sheltering Arms students were transferred to the University when their Schools of Nursing were closed.

Married.

Dr. Virgil O. Choate, Galax, Va., and Miss Millie S. Gilbert, Patrick Springs, Va., April 18th.

Dr. Clarence Porter Jones, Jr., formerly of Newport News, Va., and Miss Selma Schrunk, of Sacramento, Calif., April 25th. Dr. Jones is a member of the class of '34, Medical College of Virginia, and is at present interning at the Southern Pacific General Hospital, San Francisco. On July 1st he goes to Mater Misericordiae Hospital, Sacramento, as House Surgeon, and on October 1, 1936, to Chicago for a residency in obstetrics and for graduate work in the University of Chicago.

Dr. John A. Payne, III, Medical College of Virginia, class of '33, who interned at St. Vincent's Hospital, Norfolk, and Miss Mary Mundy, April 30th. They will be located at Sunbury, N. C., where Dr. Payne will be engaged in general practice.

Dr. Thomas Archer Gibson, Winchester, Va., and Miss Helen Hawke, Clifton Heights, Pa., in May, 1935, at Radnor, Pa.

Dr. Thomas F. Wheeldon,

Richmond, Va., was recently elected secretary-treasurer of the Harvard Club of Virginia, at its meeting in this city.

Mead Johnson Vitamin A Research Award.

In January, 1932, Mead Johnson & Company announced an award of $15,000 to be given to the investigator or group of investigators producing the most conclusive research on the vitamin A requirements of human beings. Judges for the award are: Issac A. Abt, Northwestern University; K. D. Blackfan, Harvard University; Alan Brown, University of Toronto; Horton R. Gasparis, Vanderbilt University; H. F. Helmholz, Mayo Clinic; Alfred F. Hess, Columbia University; E. V. McCollum, Johns Hopkins University; L. B. Mendel, Yale University; L. T. Royster, University of Virginia; and Robert A. Strong, Tulane University.

In February, 1933, the Judges decided on an additional award of $5,000 for a laboratory investigation which may have been published that will point the way toward clinical evaluation of the subject.

At a meeting in April, 1935, the Judges decided to postpone until December 31, 1936, awarding of the main (Clinical) award, and to divide the second (Laboratory) award, one-half to Dr. S. B. Wolbach, Harvard University, and one-half to Dr. Karl E. Mason, Vanderbilt University for their original work.

Doctors on Committees.

Dr. W. M. Phipps and Dr. D. Lane Elder, Hopewell, Va., have been appointed by the president of the Hopewell Chamber of Commerce as members of committees of that organization.

Brooklyn-Long Island Doctors Visit Richmond.

Fifty-one members of the Brooklyn-Long Island Chapter of the American College of Surgeons visited Richmond on Saturday, May 11th. Operative Clinics were held for the visitors at St. Luke's, St. Elizabeth's, Memorial, and St. Philip Hospitals, and also a Dry Clinic in the Egyptian Building of the Medical College of Virginia. After this, luncheon was served in Cabaniss Hall which is connected with Memorial Hospital.

In the afternoon clinical lectures and talks were delivered in the Auditorium of the Richmond Academy of Medicine Building, following which there was a tea for the visitors at the residence of Dr. and Mrs. J. Shelton Horsley in Westmoreland Place.

In appreciation of the program arranged by the local doctors, the visiting surgeons entertained with a dinner at the John Marshall Hotel to which were invited Dr. W. T. Sanger, President of the Medical College of Virginia; Dr. Fred P. Fletcher, President of the Richmond Academy of Medicine; and all of the local men appearing on the program. Dr. J. E. Jennings, of Brooklyn, was toastmaster, and appropriate talks were made by Drs. J. Shelton Horsley, Sanger, and Carrington Williams.

Honor for Dr. Fitzgerald.

Dr. J. O. Fitzgerald, Jr., Richmond, Va., was recently elected to membership in the American Congress of Physical Therapy.

Medical College of Virginia News.

The following men have been appointed to the faculty of the college:

Dr. A. M. Ambrose, Associate in Pharmacology.

Dr. E. L. McQuade, Associate in Preventive Medicine.

Dr. W. A. Browne, Instructor in Preventive Medicine.

Dr. T. Stanley Meade, Instructor in Pediatrics.

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Dr. L. S. Meriwether, Instructor in Neuropathology and Neurological Surgery.

Dr. P. Harrison Picot, Instructor in Gynecology.

Dr. Charles R. Robins, Jr., Instructor in Gynecology.

Dr. Vernard Benn, Assistant in Pediatrics.

Dr. Nathan Bloom, Assistant in Medicine.

Dr. E. O. Coleman, Assistant in Surgery.

Dr. Karl M. Lippert, Assistant in Surgery.

Dr. W. T. Oppenheimer, Jr., Assistant in Radiology.

Dr. L. L. Shamburger, Assistant in Obstetrics.

Dr. L. B. Todd, Assistant in Ophthalmology and Otolaryngology.

Promotion from instructors to associates were as follows:

Dr. James P. Baker, Jr., Associate in Medicine.

Dr. A. Stephens Graham, Associate in Surgery.

Dr. Guy Horsley, Associate in Surgery.

Dr. W. R. Southward, Associate in Surgery.

Dr. Rudolph Thomason, Associate in Ophthalmology.

Dr. W. T. LaNeave, Jr., was promoted from assistant to instructor in surgery, and Dr. C. W. Morhart, school of dentistry, was promoted from instructor to associate in prosthetic dentistry.

The regular scientific monthly meeting of the faculty and staff was held May 9th with the following program:

Production of Experimental Arthritis in Animals—by Dr. J. C. Forbes and Mr. R. C. Neal.

The Chemical Structures of the Hormones—by Dr. Sidney S. Negus.

Dr. C. C. Coleman reported on three cases: Colloid Cyst of the Third Ventricle, Cerebral Angiogram for Diagnosis of Intracranial Aneursym, and Neumococcic Meningitis from Petrous Abscess.

The April report for the out-patient department shows 5,565 patient visits, made by 2,575 individual patients.

Commencement exercises of the 97th session of the College were held the last of May. A report on these, with a list of medical graduates and hospital appointments will appear in the July issue of the MONTHLY.

News from University of Virgina, School of Medicine.

At the initiation meeting of the University of Virginia Chapter of Sigma Xi on April 24th the eleventh annual award of the President and Visitors' Research Prize of $100 was given to Dr. Alfred Chanutin, Professor of Biochemistry, for his paper "Experimental Renal Insufficiency Produced by Partial Nephrectomy."

On April 29th, Dr. Gunnar Nyström, of Uppsala University, Sweden, visited the Medical School.

For the second time the New York Academy of Medicine has awarded the Edward N. Gibbs Prize to Dr. Alfred Chanutin. The Prize has the value of $700 and must be used for studies on renal function.

On May 14th, Dr. H. B. Mulholland, Dr. J. Edwin Wood and Dr. Alfred Chanutin conducted a Symposium on Kidney Function and Disease before the Richmond Academy of Medicine.

On May 2nd to 4th, Dr. D. C. Smith attended the annual meeting of the American Dermatalogical Association at White Sulphur Springs, W. Va. Dr. Smith was elected to the vice-presidency of the Association for next year.

The graduation exercises of the University of Virginia Hospital Training School for Nurses were held on the afternoon of May 9th. Fifty-eight nurses received their diplomas. The address to the graduates was given by Professor Frank A. Geldard, who spoke on the subject of The Influence of Suggestion.

The Fifteenth Postgraduate Clinic conducted by the University of Virginia Hospital on May 10th was attended by forty-six physicians.

On May 9th, Dr. H. B. Stoner, of Burroughs Wellcome and Co., spoke to the fourth year class on Opportunities in Medical Research.

Dr. Karl Meyer, Director of the Hooper Foundation and Professor of Bacteriology in the Medical School of the University of California, visited the Medical School on May 13th.

News from Duke University School of Medicine.

During the month of April, Dr. Angus McBryde, Assistant Professor of Pediatrics, exchanged with Dr. Katherine Dodd, Associate Professor of Pediatrics at Vanderbilt University School of Medicine.

On April 2nd, Dr. R. A. Ross addressed the Parent-Teacher's Association of Efland, N. C., on the subject of Maternal Care, and on May 13th, he addressed the meeting of the Ladies Auxiliary of the American Legion of Durham on the same subject.

On April 9th, Dr. Bayard Carter gave a paper on The Non-Convulsive Toxemias of Pregnancy before the Eighth District Medical Society which met at Asheboro, N. C.

On April 24th, Dr. E. K. Marshall, Jr., Professor of Pharmacology and Experimental Therapeutics gave a lecture in the hospital amphitheatre on Oxygen Therapy.

At the meeting of the Durham-Orange County Medical Society in the hospital amphitheatre April 26th, Dr. Richard Kovacs, of New York City, gave an illustrated lecture on The Present Status of Physical Therapy in the Practice of Medicine.

On April 27th, Dr. Seale Harris, of Birmingham, Ala., gave a clinic in the hospital amphitheatre on Diabetes.

On May 6th, Dr. Wm. T. Davis, Professor of Ophthalmology at the George Washington University School of Medicine, gave a clinic in the amphitheatre on Refractive and Non-Refractive Asthenopia.

On May 6th, Dr. J. P. Pratt, Obstetrician and Gynecologist-in-Chief of the Henry Ford Hospital, Detroit, gave an operative clinic in the amphitheatre and a formal clinic on May 7th. His paper was on Recent Studies of the Human Corpus Luteum.

On May 6th, Dr. Bayard Carter spoke on What Constitutes Adequate Maternity Care before the Ladies Auxiliary of the American Legion at Oxford, N. C.

On May 8th, Dr. R. A. Ross gave a paper on The Paradoxes of Eclampsia before the Section of Obstetrics and Gynecology at the North Carolina State Medical Society meeting at Pinehurst, N. C.

Dr. Charles R. Robins, Jr.,

Has recently returned to Richmond, Va., and has been elected a member of the Surgical Staff of Stuart Circle Hospital. He graduated from Medical College of Virginia in 1929 and served an internship at Stuart Circle Hospital, this city, in 1929-30, after which he attended courses under Prof. Dr. Beitshe in the University of Graz in Austria. Returning to the States, Dr. Robins had a fellowship in surgery at the Mayo Clinic for 1931-34, and for the past year has been first assistant to Dr. Charles W. Mayo.

Dr. William A. Farmer,

Formerly a resident physician at the Medical College of Virginia Hospital, Richmond, later going to the American Hospital in Paris, has returned and has located at Anderson, S. C., where he will be engaged in general surgery.

Dr Philip E. Rossiter,

In 1930 and 1931 medical officer of the James River Fleet, with headquarters at Lee Hall, Va., later going to Chemung, N. Y., has now been transferred to the Veterans Hospital, Bath, N. Y.

Dr. William F. Bryce, Jr.,

Class of '34, Medical College of Virginia, who has been serving an internship at the Medical College of Virginia Hospitals, Richmond, has received an appointment in ophthalmology at the Mayo Clinic, and will enter upon his new duties on July 1st.

Dr. C. C. Freed,

Waynesboro, Va., was elected president of the Rotary Club of that city, at the meeting early in May.

Dr. D. E. Watkins was program chairman for this meeting.

The New Veterans' Hospital,

Roanoke, Va., was opened for the reception of patients on April 23, 1935. This hospital is primarily a hospital for the treatment of nervous and mental disorders. A limited number of beds is available for the reception of veterans eligible for, and in need of emergency hospitalization for surgical and medical disabilities.

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Physicians throughout the state are kindly requested not to refer veterans to this hospital without first ascertaining that a bed is available and that eligibility of the veteran to receive treatment has been established.

The medical staff consists of the following medical officers: Dr. L. E. Trent, Chief Medical Officer; Dr. C. T. Wilfong, Chief, Out-Patient Clinic; Dr. R. L. Eltinge, Dr. Paul M. Jerrell, Dr. H. B. West, Dr. D. P. Stearns, Dr. Lucian C. Smith, Dr. Fred F. Oast, Dr. C. E. Clay, Dr. H. W. Kennard, Dr. E. M. Hasbrouck, Dr. J. E. Neilon.

The following named physicians have been appointed as Consulting Specialists: Dr. Hugh S. Trout, Roanoke, Va., Consulting Surgeon; Dr. G. M. Maxwell, Roanoke, Va., Consulting Eye, Ear, Nose and Throat Specialist; Dr. J. T. McKinney, Roanoke, Va., Consulting Roentgenologist; Dr. L. D. Keyser, Roanoke, Va., Consulting Surgeon; Dr. W. L. Powell, Consulting Internist; Dr. Churchill Robertson, Roanoke, Va., Consulting Internist; Dr. John O. Boyd, Roanoke, Va., Consulting Internist; Dr. Thos. N. Spessard, Roanoke, Va., Consulting Neuropsychiatrist; Dr. D. P. Scott, Lynchburg, Va., Consulting Internist; Dr. S. Beverly Cary, Roanoke, Va., Consulting Specialist in Genito-Urinary Diseases; Dr. Hugh Hagan, Roanoke, Va., Consulting Internist.

Dr. W. Fredric Delp,

Of the '34 class, Medical College of Virginia, who interned at the University Hospital, Ann Arbor, Mich., is opening offices for general practice in the Crowell Building, Pulaski, Va.

Dr. Clarence Porter Jones,

Newport News, Va., addressed the Eye, Ear, Nose and Throat Section of the Norfolk County Medical Society, April 29th. His subject was "Some Problems of Mutual Interest to Specialists and Practitioners."

Foods and Nutrition.

An interesting and instructive booklet on food values and nutrition, covering particularly the newer knowledge of vitamin and mineral metabolism, has been prepared by the R. B. Davis Company, the makers of Cocomalt. Charts showing the relative content of vitamins, calcium, phosphorus, and total caloric value of the most common foods are presented.

This may be secured without cost on request to R. B. Davis Company, Dept. 000, Hoboken, N. J.

The Gill Memorial Eye, Ear and Throat Hospital

Recently completed its Ninth Annual Spring Graduate Course with a total registration of sixty-five doctors. Sixty doctors were refused admission due to lack of accommodations.

The following visiting doctors delivered lectures:

Dr. Oscar V. Batson, Philadelphia, Pa.
Dr. William V. Mullin, Cleveland, Ohio.
Dr. Lyman Richards, Boston, Mass.
Dr. John A. Kolmer, Philadelphia, Pa.
Dr. Harry S. Gradle, Chicago, Ill.
Dr. Ben Witt Key, New York City, N. Y.
Dr. Ralph I. Lloyd, Brooklyn, N. Y.
Dr. A. B. Reese, New York, N. Y.

The program next year will be given the second week in April.

Dr. C. Curtis Hatfield,

Class of '34, Medical College of Virginia, who interned at St. Vincent's Hospital, Erie, Pa., has located at Saltville, Va., where he will engage in general practice.

Army Doctors Transferred.

Dr. C. L. Neale, recently at Tripler General Hospital, Honolulu, Hawaii, has been transferred to Fort Sam Houston, Texas, where he will be engaged in gynecological and obstetrical work. He has recently been visiting his former home in Virginia.

Dr. C. L. Brothers, who was stationed at Schofield Barracks, Hawaii, has been transferred to Kelly Field, Texas, for duty.

Dr. John R. Wood, who has been at Manila, P. I., has been ordered to Walter Reed General Hospital, Washington, D. C.

All three of these doctors are members of the class of '28, Medical College of Virginia, and are now captains in the Medical Corps of the U. S. Army.

Dr. W. N. Breckinridge,

Fincastle, Va., was recently re-elected chairman of the Botetourt County Democratic committee.

Dr. James S. Dryden,

Recently of Jeffs, Va., has moved to Oakland, Md,. having been appointed as physician to the CCC Camp at that place. He is a member of

the class of '33, Medical College of Virginia and later interned at St. Vincent's Hospital, Norfolk, Va.

Dr. Bernard C. Grigsby,

Class of '33, Medical College of Virginia, who has recently been located at Quinwood, W. Va., has moved to Bonny Blue, Va., where he is associated with Dr. C. H. Henderson.

Dr. T. R. Boling,

Class of '31, Medical College of Virginia, recently of Grantsville, W. Va., has been appointed acting surgeon with the U. S. Public Health Service. He entered upon his new duties on May 5th, at the U. S. Quarantine Station, San Juan, Porto Rico.

Dr. F. T. Renick,

Recently of Crozet, Va., has located at Grantsville, W. Va., for general practice. He graduated from the Medical College of Virginia in 1931.

The Southside Virginia Medical Association

Is to hold its regular quarterly meeting in Lawrenceville, on Tuesday, June 11th. Dr. S. E. Gunn, Hopewell, is president, and Dr. R. L. Raiford, Franklin, secretary.

Dr. C. P. Ryland,

Binghamton, N. Y., is in New York City, where he is taking a four weeks' seminar in internal medicine at the New York Postgraduate Medical School. Dr. Ryland is an alumnus of the Medical College of Virginia and was located at Buena Vista, Va., before going to Binghamton.

Dr. and Mrs. Paul L. Dent,

Recently of Point Pleasant, W. Va., left the latter part of May for Keams Canyon, Ariz., where Dr. Dent will be with the Hopi Indian Agency.

Dr. C. Howard Cain,

For some time of Orange, Va., is now in Conway, S. C. Dr. Cain is a member of the class of '30, Medical College of Virginia.

Dr. D. F. Love,

For the past three and a half years engaged in industrial practice at Raysal, W. Va., left that place with his family on the first of June for a month's vacation before going to the Children's Hospital, Pittsburgh, Pa., where he has been accepted for a residency in pediatrics. Dr. Love is an alumnus of the Medical College of Virginia, class of '28, and, before going to West Virginia, was located at New Market, Va.

Dr. F. B. Teague,

For a time health officer of Martinsville, Va., has just resumed the private practice of medicine in that city.

Dr. A. T. Brickhouse,

Formerly of Hopewell, Va., has returned to that place and resumed his work as assistant to Dr. D. Lane Elder, after about a year's service at the CCC Camp at Fort Story.

Heads State Pharmacy Board.

J. Conrad Kearfott, of Martinsville, Va., has been elected president of the Virginia State Pharmacy Board for the coming year. A. L. I. Winne was re-elected secretary-treasurer.

Dr. D. O. Hamblin,

An alumnus of the University of Virginia School of Medicine, class of '29, recently of Pennington Gap, Va., is now located at Bound Brook, N. J.

Dr. Byrd C. Willis,

Rocky Mount, N. C., was elected president of the North Carolina Hospital Association, at the joint meeting of the Hospital Associations of Virginia, North and South Carolina, held recently in Greensboro, N. C.

The Graduate Nurses' Association of Virginia,

At its annual session in Charlottesville in May, elected the following officers for the ensuing year: Vice-presidents, Miss Elizabeth Buxton, Roanoke, and Miss Mary Mastin, Richmond; secretary, Miss Mary E. Marshall, Hampton; and treasurer, Mrs. Jessie W. Faris, Richmond. Miss Frances Helen Zeigler, Richmond, continues as president, having been elected in 1934 for a two-year term.

Warning!

Doctors are warned against a stranger who gains admission to their offices and secures information about them. He obtains prescription forms, blank checks, etc., and uses them as a means of identification in buying supplies and having checks cashed in the doctor's name. Recent cases have been reported in Virginia and North Carolina.

The description given in two cases is that of a man about 45 years of age, 5 feet, 8 inches tall, about 160 pounds in weight, ruddy complexion, and dark hair which is slightly gray at the tem-

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ples. He is very familiar with surgical instruments and supplies.

The American Public Health Association

Announces the appointment of Reginald M. Atwater, M. D., Dr. P. H., as Executive Secretary of the Association. For the past eight years he has been Commissioner of Health in Cattaraugus County, New York. Dr. Atwater was formerly Associate Professor of Hygiene in the Hunan-Yale College of Medicine in Changsha, China, from which post he returned to the United States in 1925 to teach in the Harvard School of Public Health. Dr. Atwater is the author of several studies in epidemiology.

Graduate Fortnight for 1935.

The New York Academy of Medicine will hold its eight annual Graduate Fortnight October 21st to November 2nd. This will be devoted to a consideration of Diseases of the Respiratory Tract. Eighteen important hospitals of the city will present coordinated afternoon clinics and clinical demonstrations. At the evening meetings prominent clinicians from various parts of the country, recognized authorities in their special lines, will discuss various aspects of the general subject. There will also be a comprehensive exhibit of books and of anatomical, bacteriological and pathological specimens and research material.

The profession generally is invited to attend. Those interested may obtain a complete program and registration blank from Dr. Frederick P. Reynolds, The New York Academy of Medicine, 2 East 103rd Street, New York City.

The Leslie Dana Gold Medal,

Awarded annually for outstanding achievements in the prevention of blindness and the conservation of vision was presented this year to Dr. William H. Wilder, of Chicago. Dr. Wilder was selected for this honor by the National Society for the Prevention of Blindness in cooperation with the St. Louis Society for the Blind. The presentation ceremonies took place in St. Louis on May 18th. The medal bears the inscription: "Wise Clinician, Devoted Teacher and Humanitarian."

Dr. Wilder is secretary-treasurer of the American Board of Ophthalmology; professor emeritus of ophthalmology at Rush Medical College, University of Chicago; and a past president of the American Academy of Ophthalmology and Otolaryngology.

For Sale—

Model 707 McKessen gas machine. Unused and a bargain. Address No. 486, care the VIRGINIA MEDICAL MONTHLY, 1200 East Clay Street, Richmond, Va. (*Adv.*)

Obituary Record

Dr. Luther Addison Robertson,

Prominent eye, ear, nose and throat specialist of Danville, Va., died May 16th at a Richmond hospital, from embolism several days after an operation for hernia. Dr. Robertson was a native of Pittsylvania County, Virginia, and was 61 years of age. He studied for his profession at the former University College of Medicine, Richmond, from which he graduated in 1900, and was assistant to Dr. J. A. White of this city for a time before locating in Danville. Besides being affiliated with many other organizations, he had been a member of the Medical Society of Virginia since 1901.

Dr. Robertson was unmarried but is survived by his sister and three brothers, one of these being Dr. W. W. Robertson, of Danville.

Dr. Blanton L. Hillsman,

Surgeon of the Police Department of Richmond, Va., died in his office on May 17th as the result of a heart attack. Dr. Hillsman was 63 years old and a native of Amelia County, Virginia. He was graduated from the former University College of Medicine, Richmond, in 1898 and had been practicing in this city since that time.

Dr. Hillsman, who served overseas in the World War, held the rank of colonel at the time of his discharge. He was a member of the Richmond Academy of Medicine as well as several fraternal and civic organizations. He is survived by his wife, two sons, and a daughter.

Dr. Jesse L. McElroy,

From 1929 to 1933, superintendent of the hospital division of the Medical College of Virginia, died as the result of a heart attack on May 5th. Dr. McElroy, who was 50 years of age, was a native of Roelsville, Ind., and attended the Indiana Medical College from which he was graduated in 1907. He was widely known as a hospital executive, at the time of his death, being director of the American Hospital, Neuilly-Sur-Seine, France. He is survived by his wife and a son.

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